



*The Management of*  
EMERGENCIES IN  
THORACIC SURGERY

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APPLETON CENTURY-CROFTS, INC  
NEW YORK

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Library of Congress Card Number 58-6553

578-1

PRINTED IN THE UNITED STATES OF AMERICA

*To My Wife*

HELEN





## Preface

Emergencies in thoracic surgery range widely from cardiac arrest to ruptured esophagus and from bilateral spontaneous pneumothorax to strangulated diaphragmatic hernia. Their common pathologic link is that they all create a state of emergency which must be promptly recognized and treated if satisfactory results are to be achieved.

Much of what has been written on thoracic emergencies still lies scattered widely throughout an extensive literature and has not yet been brought together in textbook form. Yet the knowledge of how to recognize the emergency, its dangerous points and what to do is so important that the time has come for a monograph on the subject.

There are few groups of physicians who do not meet such emergencies at some time in their medical career be they general practitioners faced with an endobronchial foreign body masquerading as acute asthma, residents or ambulance doctors faced with crush injuries of the chest or spontaneous rupture of the esophagus disguised as coronary thrombosis obstetricians faced with hydramnios and babies that drool saliva because of a tracheoesophageal fistula, pediatricians faced with problems of acute laryngeal edema or tension pyothorax in babies physicians faced with severe hematemesis from ruptured esophageal varices, residents watching spontaneous hemopneumothorax exanguinate their patients psychiatrists faced with problems of corrosive burns of the esophagus, or finally, the anesthetist or surgeon faced with cardiac arrest.

The aim of this book is to present those thoracic lesions which are essentially surgical emergencies and to describe their pathologic nature their clinical features and their management. Those who are familiar with thoracic surgery know full well that many standard operations such as thoracoplasty, lobectomy pneumonectomy and esophageal anastomosis can also have postoperative complications which are themselves real emergencies. They too will be briefly described.

This book will help general practitioners house staff, and consultants appreciate the emergencies of a new specialty from the surgeon's viewpoint. They usually see the patient before the surgeon is called and should be aware of what surgery can offer. Under present hospital practice it is usually an hour or more before the patient is in the operating room. Therefore the faster the state of emergency is recognized and the surgeon consulted, the sooner these and other delays will be shortened.

This book will introduce medical students to lesions which they will meet, often unexpectedly throughout their professional lives and which they might otherwise find baffling.

I am happy to acknowledge initial encouragement in the writing of this book from Professor Howard C. Hopps, Department of Pathology University of Texas, Galveston, who spent his sabbatical leave of 1955 at the University of Otago Medical School New Zealand. Through him I was introduced to Mr. George A. McDermott, the Medical Editorial Director at Appleton-Century-Crofts, Inc. and to the

Hooper, and also the enthusiastic support of Miss Catherine Entwistle, Medical Artist For photographs, I am indebted to Mr Gerald Brooks, Photographic Unit, Otago Medical School, and also to Mrs Daphne Lemon and Mr Kenneth White I thank Mr Litherland, Department of Photography, Auckland Hospital, for other photographic work done during my term at Green Lane Hospital

In the matter of references to published literature, Miss E Murray of the Medical Library gave great assistance, as did Mr H D Erlam, the chief librarian Dr Robert Christie read the manuscript with great care and Dr Graham Campbell assisted me in reading the page proof Their willing help is gratefully acknowledged

Finally, I humbly thank my wife for all her patient encouragement, for her reading of drafts and manuscript, and for her cheerful shouldering of the greater share of caring for our young family during the time of writing

JOHN BORRIE

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## Foreword

The relative newness of thoracic surgery as a speciality is reflected in the comparative paucity of standard textbooks on the subject. The newly appointed resident or registrar may not wish to plough through a full length book in order to learn about the imminent and more practical problems with which he will be faced. A formal textbook often has to devote so much space to a comprehensive presentation of all aspects of the subject that inevitably the practical everyday things tend to receive less attention. This seems to have guided Mr Borrie in his presentation of the subject of the management of emergencies in thoracic surgery. Although true emergencies are dealt with, the book covers more than these, it includes all the everyday manipulations and procedures which form the basis of so much of the routine of thoracic surgery.

Today thoracic surgery is becoming less of a closed speciality, the general surgeon has expanded through the diaphragm into the chest and the whole field of chest injuries may also involve him. He and his team must be familiar with the basic anatomical and physiological factors involved in chest wounds or chest operations and their clinical application. Correct preoperative and postoperative management is essential to success and to lower morbidity and mortality. This management is clearly and simply presented by Mr Borrie with a wealth of illustration that makes for absolute clarity and simplicity. Those dealing with thoracic cases, whether senior or junior, will find a description of all the various manipulations and manoeuvres they may be called upon to use. Especially valuable is the account of such features as sputum retention and its prevention and treatment, of pleural drainage and the varieties and perils of pneumothorax. In no other book that I know is this practical side of thoracic surgical work presented so comprehensively and so lucidly.

The preoperative and postoperative care of chest patients involves others in addition to surgeons. The nursing staff, the physiotherapists and other ancillaries will have no difficulty in profiting from this book, so clearly is it written.

In such a wide subject there are bound to be some variations of opinion and of teaching, and one inevitably finds features with which one is not wholly in agreement. This is not to say that alternative views are not correct; they may in fact be more correct and more acceptable. In every instance Mr Borne explains his recommendations by giving the factors which guide him.

I wish him every success in this book which should find a wide field of readers, both senior and junior specialist and nonspecialist, who will profit and learn from it and will acquire confidence and knowledge of great value to their practical surgical chest work.

RUSSELL BROCK



## BASIC PRINCIPLES OF THORACIC SURGERY

**Introduction** An understanding of the principles of thoracic surgical anatomy and physiology is as fundamental as a knowledge of pathology in recognizing and appreciating all the problems of the management of thoracic operations

Historically the generation of surgeons who followed Lister's (1) emancipation of surgery in 1867 was not slow to appreciate the surgical possibilities of thoracic disease but their operations based on pathology and general surgical technic alone almost invariably failed, because of insufficient regard for those equally important principles of thoracic physiology. Postoperative sputum retention, atelectasis, pneumothorax, and pleural infection were all rocks on which their ventures foundered.

Although anesthesia, blood replacement and antibiotic therapy have all contributed to the rapid progress of the past 20 years with careful surgical technic, blood loss can often be minimized and the need for antibiotic prophylaxis against infection much reduced or even made unnecessary. Success today is achieved even more than by perfection of technic by the frequent and painstaking assessment of chest function for at least two weeks following operation and the prompt correction of any errors that become manifest.

An essential prerequisite for treating thoracic emergencies is that the potential of thoracic surgery be recognized by all persons handling such problems and that the emergency be promptly diagnosed and referred for surgical opinion.

Success is equally dependent on the teamwork of keen residents and house staff, a department of radiology prepared to take high quality chest x-ray films at all times both night and day and a competent operating-theater staff. Also needed is a team of nurses and physiotherapists who understand the aims of the surgeon and who by striving for the return of full function to the lungs give the patient further confidence and encourage his convalescence.

**Fundamental Principles.** The fundamental fact to remember in the surgical phase of treating chest lesions is that the lungs in fulfilling their role of transferring oxygen from the air to the blood stream for tissue oxidation, require

- (a) A clear airway from the mouth via the bronchial tree to the alveoli of the lung
- (b) Positive pressure inflation of the lungs during operation
- (c) Full re-expansion of the lung by removal of air and fluid from the pleural cavity after operation
- (d) A soundly reconstituted chest wall
- (e) Normal blood volume

As Johnson and Kirby (2) stress, "The thoracic surgeon must be trained to think constantly in terms of function." To understand these points better and to know how when and why to correct them when disordered require an understanding of the mechanics of the bronchi and pleura, which will now be discussed.



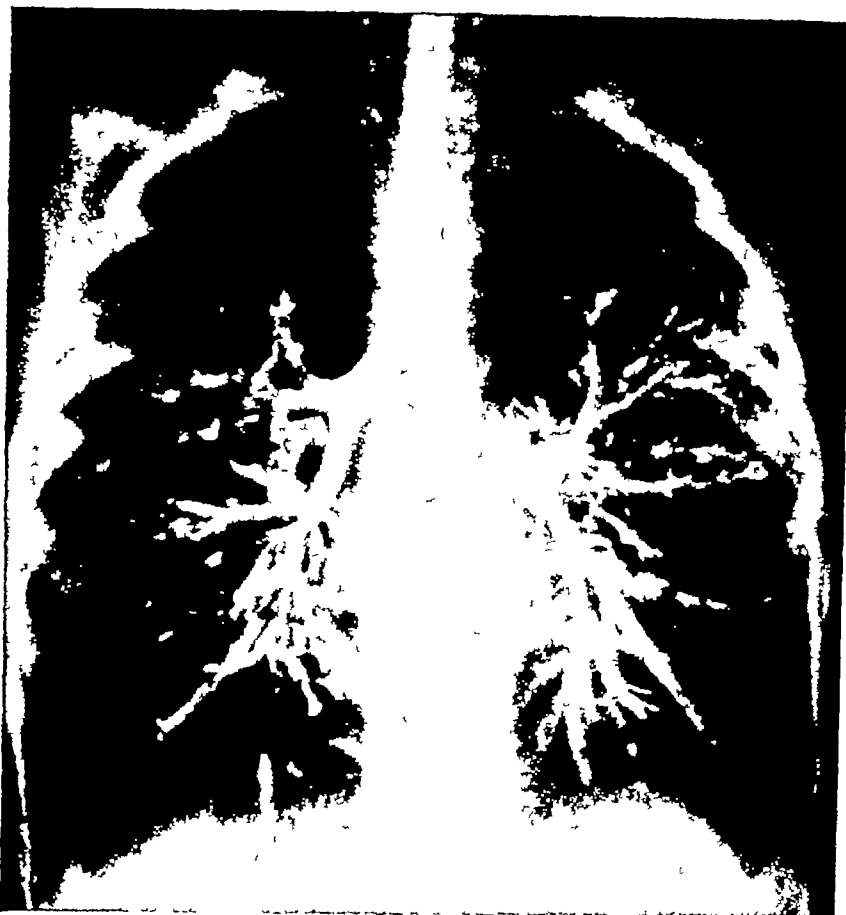


Fig 1A Bronchogram showing normal outline of the bronchial tree

(The regional anatomy and surgical approach to the thoracic cavity are ably described by Sweet (3) and will not be considered here )

### ✓SURGICAL ANATOMY OF THE AIRWAY

The practitioner need hardly be reminded that the respiratory mechanism includes not only the airway—comprising the nose, pharynx, larynx, trachea, and lungs with their bronchi, bronchioles and terminal alveoli—but also the diaphragm and the thoracic and abdominal walls. It must also be borne in mind that, from the viewpoint of the thoracic surgeon, the two parts of the airway commanding the greatest respect are the vocal cords and the bronchi.

**The Vocal Cords.** Situated in the larynx, the vocal cords, under certain conditions, may obstruct the airway either totally or in part when:

- (a) The left cord is paralyzed from direct neoplastic invasion of the left recurrent laryngeal nerve or from external pressure from an intrathoracic neoplasm or aneurysm,
- (b) A foreign body such as bone or eggshell becomes wedged between the cords;
- (c) Edema of the glottis not only forms an effective obstruction to normal respiration but also prevents expulsion of intrapulmonary secretions

**The Bronchi.** The need for accurate diagnosis and treatment of pulmonary disease in terms of the bronchial tree led to widespread study and a clearer description of the segmental anatomy of the lung, culminating in 1946 in the publication of Brock's *The Anatomy of the Bronchial Tree* (4). In 1949, at the International Con-

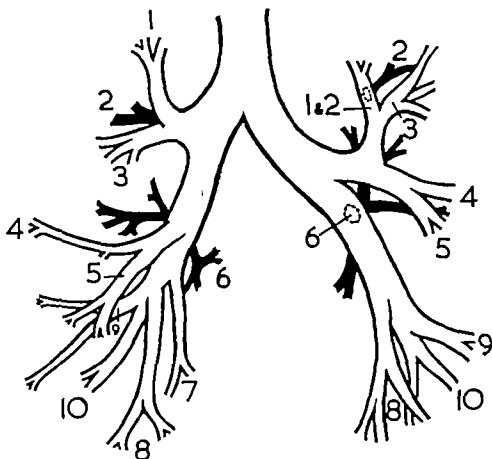


Fig. 1A. (cont) Diagram of major branches of bronchial tree.

*Right lung upper lobe.* 1 apical, 2 posterior 3 anterior *middle lobe* 4 lateral, 5 medial *lower lobe* 6 apical, 7 medial (cardiac) 8. anterior basal, 9 lateral basal, 10 posterior basal. *Left lung: upper lobe* 1 apical, 2 posterior 3 anterior 4 superior lingula, 5 inferior lingula, *lower lobe* 6 apical, 8 anterior basal, 9 lateral basal, 10 posterior basal.

ference (5) agreement was reached on an acceptable nomenclature for the various bronchopulmonary segments (Fig 1) More recently the painstaking studies of Boyden (6) have further added to the detailed knowledge of the peripheral branches of the major bronchi Much of the important new knowledge of the anatomy and surgical technics of the thorax was collected in 1954 by Birnbaum (7)

The description that follows is of the major bronchi and their immediate branches which are the accessible and significant parts of the airway in the management of thoracic operations

**RIGHT MAIN BRONCHUS** The right main bronchus follows the vertical direction of the trachea more closely than the left main bronchus does The right main bronchus is shorter and wider than the left and is therefore the more common site for intrapulmonary foreign bodies

The main and segmental divisions of the right main bronchus are as follows

Right Upper Lobe Bronchus	Middle Lobe Bronchus	Right Lower Lobe Bronchi
Apical segment	Lateral segment	Apical segment
Anterior segment	Medial segment	Medial basal segment
Posterior segment		Anterior basal segment
		Lateral basal segment
		Posterior basal segment

The first branch of the right main bronchus is the *right upper lobe bronchus* This arises 1.5 cm from the trachea and lies above the main pulmonary artery The

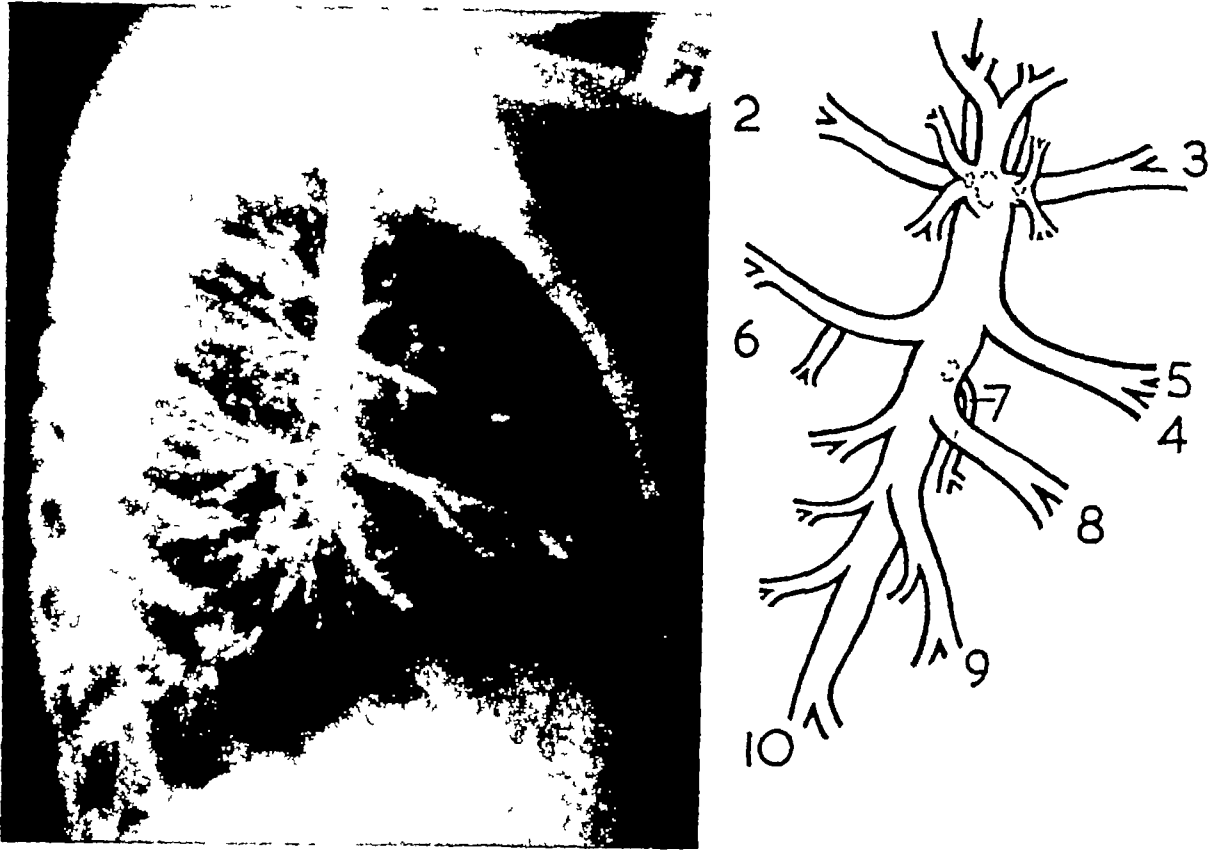


Fig 1B Lateral view of the right bronchial tree Diagram clarifies the position of its main segmental branches, whose numbers are the same as those in the legend of Figure 1A

right upper lobe bronchus passes upward and laterally from the main bronchus before dividing into *apical*, *anterior*, and *posterior* segmental bronchi

The second branch of the right main bronchus is the *middle lobe bronchus*. This arises from the ventral aspect of the main bronchus 3 cm below the upper lobe bronchus and divides into *medial* and *lateral* segmental bronchi

The third and fourth branches of the right main bronchus are the *right lower lobe bronchi*. The third branch is directly posterolateral across the main bronchus and leads to the *apical* segment of the right lower lobe. The main bronchus then descends to its fourth or terminal branch which divides into the *medial basal*, *anterior basal*, *lateral basal*, and *posterior basal* segmental bronchi

**LEFT MAIN BRONCHUS** The left main bronchus has a longer and more oblique course than the right main bronchus. The main and segmental divisions of the left main bronchus are as follows

#### Left Upper Lobe Bronchus

Apical segment  
Posterior segment  
Anterior segment

#### The Lingula

Superior segment  
Inferior segment

#### Left Lower Lobe Bronchus

Apical segment  
Anterior basal segment  
Lateral basal segment  
Posterior basal segment

The first branch of the left main bronchus is the *left upper lobe bronchus*. This arises 2.5 to 3 cm from the trachea and has the left main pulmonary artery winding around its lateral surface. The left upper lobe bronchus divides into three segmental branches: *apical*, *posterior*, and *anterior*. The lingula, which corresponds to the middle lobe branch of the right main bronchus, is fused to the anterior segment of the left upper lobe bronchus and is divided into *superior* and *inferior* segmental bronchi

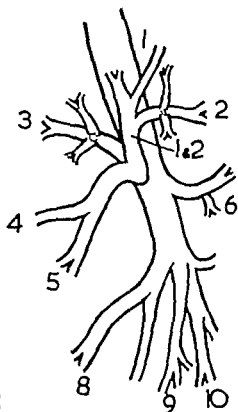
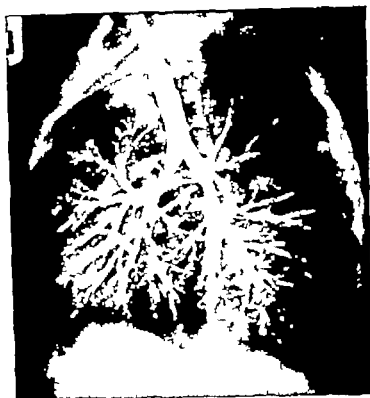


Fig. 1C. Left oblique view of the left bronchial tree. Diagram similarly clarifies the position of its main segmental branches, whose numbers are also the same as those in the legend of the preceding Figure 1A

The second branch of the left main bronchus arises dorsally just below the left upper lobe bronchus and leads to the *apical segment* of the left lower lobe. The left lower lobe bronchus divides further into *anterior basal lateral basal* and *posterior basal segmental* bronchi. There is no separate left medial basal bronchus.

**Bronchoscopic Appearances.** The bronchoscopic appearances of the trachea and bronchi follow the descriptions just given and are easily recognized (Fig. 2). Once the bronchoscope is past the vocal cords the rings of the trachea are seen arching in front with the unsupported muscular part lying behind. The tracheal bifurcation or carina is normally sharp. In the presence of disease however the carina may be widened because of enlargement of lymph nodes lying directly beneath it.

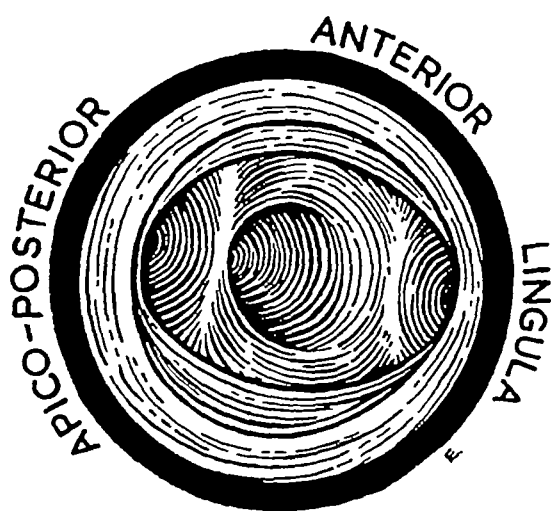
**RIGHT MAIN BRONCHUS.** The first branch on its lateral side is the right upper lobe bronchus whose three secondary bronchial orifices are clearly visible with a right angle telescope. Beyond this, and ventrally at 12 o'clock, lies the opening of the middle lobe in which are the orifices of its two major branches.

Dorsally opposite the middle lobe at 5 o'clock, is the opening of the apical segment of the lower lobe. At the end of the main bronchus three basal bronchial orifices (anterior lateral and posterior) are recognized, while just proximal to this point, and in the medial wall is the opening of the medial basal bronchus.

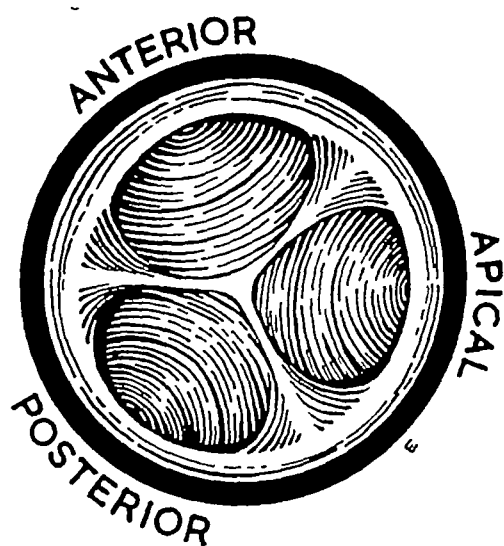
**LEFT MAIN BRONCHUS.** With the patient's head turned to the right and raised forward, the bronchoscope will readily pass to the left and beneath the aortic arch. The upper lobe orifice arises on the left side some 4 cm. from the main carina. Normally the origin of the lingula is obvious adjacent to the upper lobe carina. A right angle telescope however is required to see the origin of the remaining three secondary bronchi. The pattern of the left lower lobe corresponds to that of the right lower lobe except that the apical bronchus is subjacent to the upper lobe orifice and that there is no medial basal bronchial orifice.



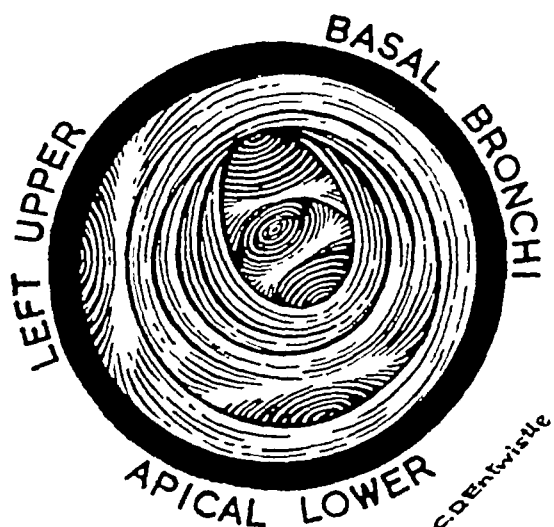
CARINA



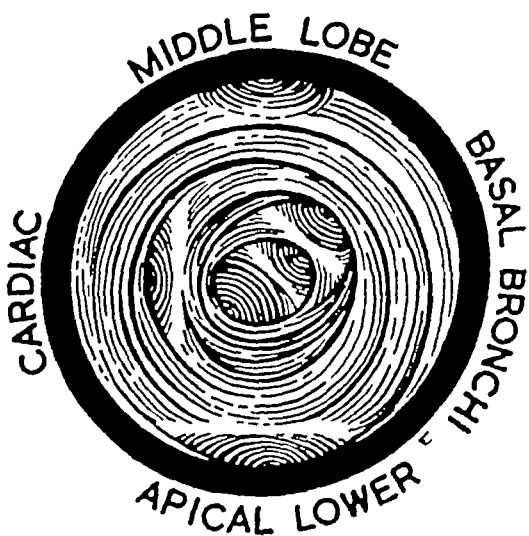
LEFT UPPER BRONCHI



RIGHT UPPER BRONCHI



LEFT LOWER BRONCHI



RIGHT LOWER BRONCHI

Fig 2 Bronchoscopic appearance of carina and main bronchi

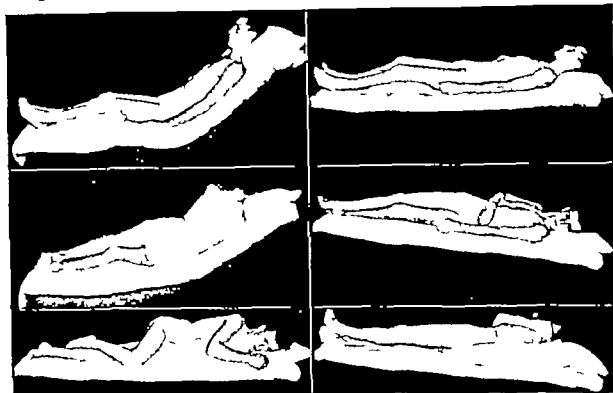


Fig. 3A. Upper lobes. Positions for postural drainage.

Top Apical segment

Middle Posterior segment, left

Bottom Posterior segment, right

Top Anterior segment

Middle Middle lobe, right lung

Bottom Lingula, left lung

**Postural Drainage.** This is determined by the anatomy of the bronchial tree and aims at providing dependent drainage for the affected bronchus. Postural drainage is of particular value in the medical and surgical treatment of bronchiectasis and lung abscess and as a means of clearing the lung of secretions before and after bronchography.

The patient assists expulsion of secretions by deep breathing and coughing under the supervision of a physiotherapist, who may further dislodge secretions by percussion or pummeling.

The required positions are illustrated in Figure 3.

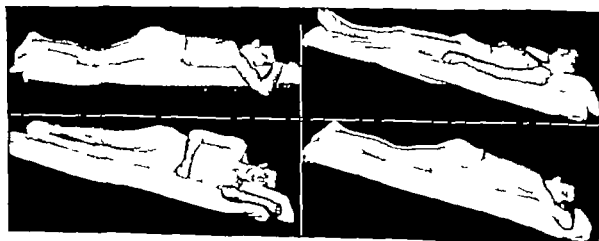


Fig. 3B. Lower lobes. Positions for postural drainage.

Top Apical segment

Bottom Right lateral basal segment

Top Anterior basal segment

Bottom. Posterior basal segment



Fig 4 Emphysema of right lower lobe in a child, displacing heart and mediastinum to left. The lower lobe bronchus was partially blocked by tuberculous peribronchial lymph nodes.

### BRONCHIAL MECHANICS AND SURGICAL PATHOLOGY

The bronchi normally dilate and elongate on inspiration and relax on expiration. This cycle of events is altered by lesions which either partially or completely obstruct the bronchial lumen and which may block a central or peripheral bronchus.

**Partial Bronchial Obstruction.** When a bronchus is partially blocked, as by a neoplasm or a foreign body, air can more readily pass in beyond the block than leave again. The affected lung segment inevitably becomes emphysematous and may cause one of three conditions, of which the first and second are common and the third is rare.

1 The overdistended portion of the lung expands, forcing the heart and mediastinum to the opposite side and compressing and embarrassing all normal lung tissue (Fig 4).

2 When one peripheral bronchus alone is affected, the ensuing localized emphysema may cause a "tension cavity" to develop in the lung parenchyma. Such cavities are seen in pulmonary tuberculosis, lung abscess, staphylococcal pneumonia, foreign body, neoplasm, or degenerative emphysema (Fig 5A).

3 Rarely, the lung or tension cavity may rupture into the pleural cavity to produce a pneumothorax (Fig 5B). If the leak is valvular, a tension pneumothorax results.



Fig. 5A. Tension cavity in left lower lobe of a baby displacing heart to right.

**Total Bronchial Obstruction.** A bronchus may be totally blocked from

- (a) *Endobronchial* causes such as neoplasm, foreign body, postoperative sputum retention, or retention of secretion behind a partial obstruction and
- (b) *Extrabronchial* causes such as pressure from a neoplasm or enlarged lymph nodes

The result is that, as air trapped in the distal bronchi and alveoli becomes absorbed into the blood stream the affected segment becomes collapsed or atelectatic.

Although by derivation the word "atelectasis" refers to lung tissue which has never been aerated, by common usage it now refers to lungs or their segments collapsed from endobronchial causes, thus leaving the word "collapse" to describe the relaxation and compression collapse of the lung parenchyma most commonly encountered with pneumothorax therapy for pulmonary tuberculosis. In this type of collapse, the bronchi are patent.

Atelectasis has an immediate adverse effect on the pleural cavity and a delayed one on the lung.



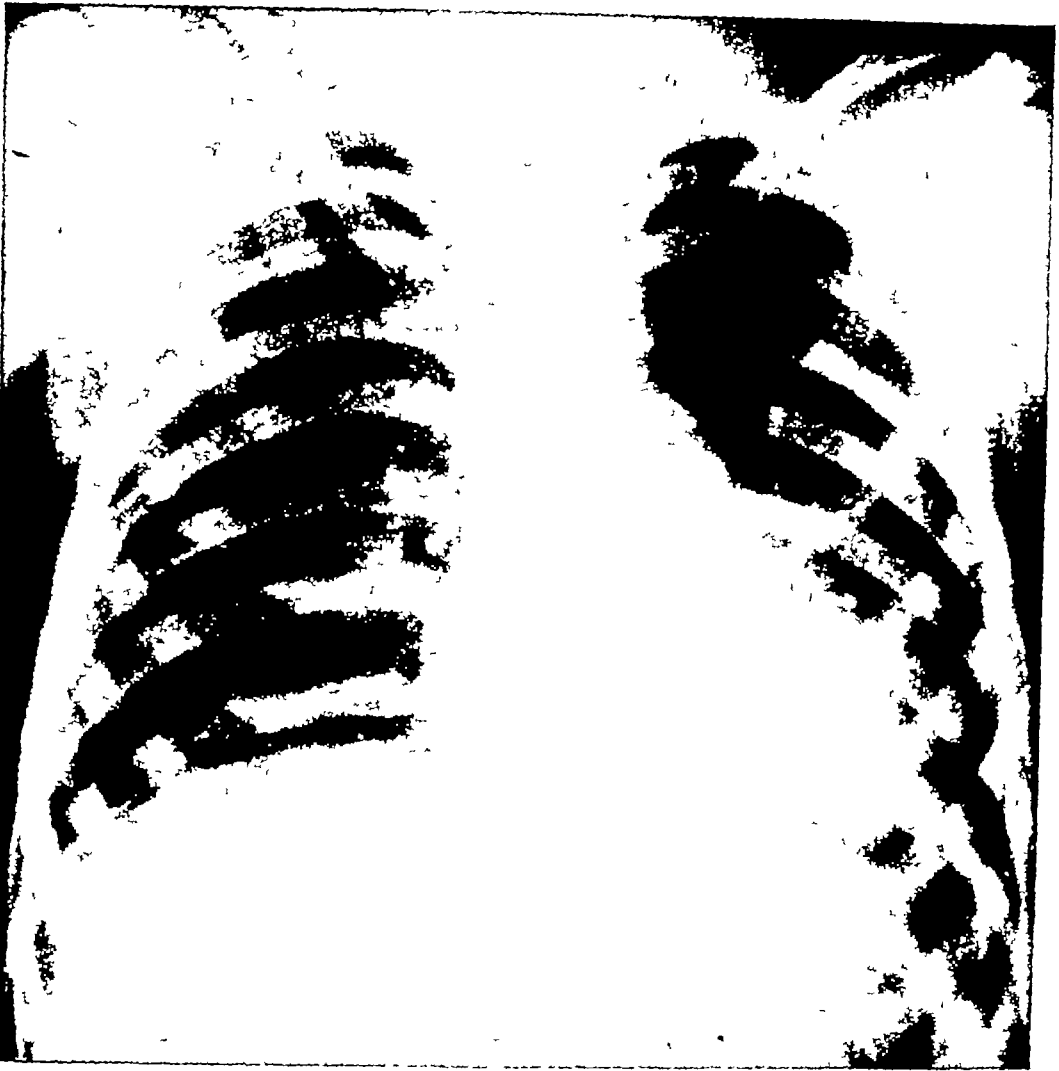


Fig 5B. Left pneumothorax in a 5-year old boy caused by peanut partially occluding left main bronchus

**EFFECT OF ATELECTASIS ON PLEURAL CAVITY.** As an atelectatic lobe or lung occupies only a fraction of the volume of its aerated state, with an accompanying fall of intrapleural pressure, the normal lung becomes relatively overdistended. Atmospheric pressure exerted *from within* through the lungs is responsible for the heart and mediastinum moving to the affected side. Exerted *from without*, atmospheric pressure is responsible for the ribs becoming approximated and the subjacent leaf of the diaphragm being raised in an attempt by nature to restore the intrapleural pressure to normal.

Lobar atelectasis produces characteristic x-ray patterns which are shown in Figures 6A-F. These must be familiar to all persons interested in the recognition and treatment of chest ailments.

**EFFECT OF ATELECTASIS ON LUNG.** Within the atelectatic area there is stasis, which inevitably leads to infection, with the result that:

- (a) The bronchi may become bronchiectatic;
- (b) The lung alveoli may become congested and pneumonic;
- (c) The lung parenchyma may become the seat of a lung abscess; or
- (d) An overlying pleural reaction may produce pleurisy with effusion and even empyema.

Such a chain of events, associated with infection, explains why carcinoma of the lung so often masquerades as lobar pneumonia, influenza, lung abscess, or even empyema.



Figs. 6A and B. X-ray pattern of lobar atelectasis. Posteroanterior and lateral views of right upper lobar atelectasis.

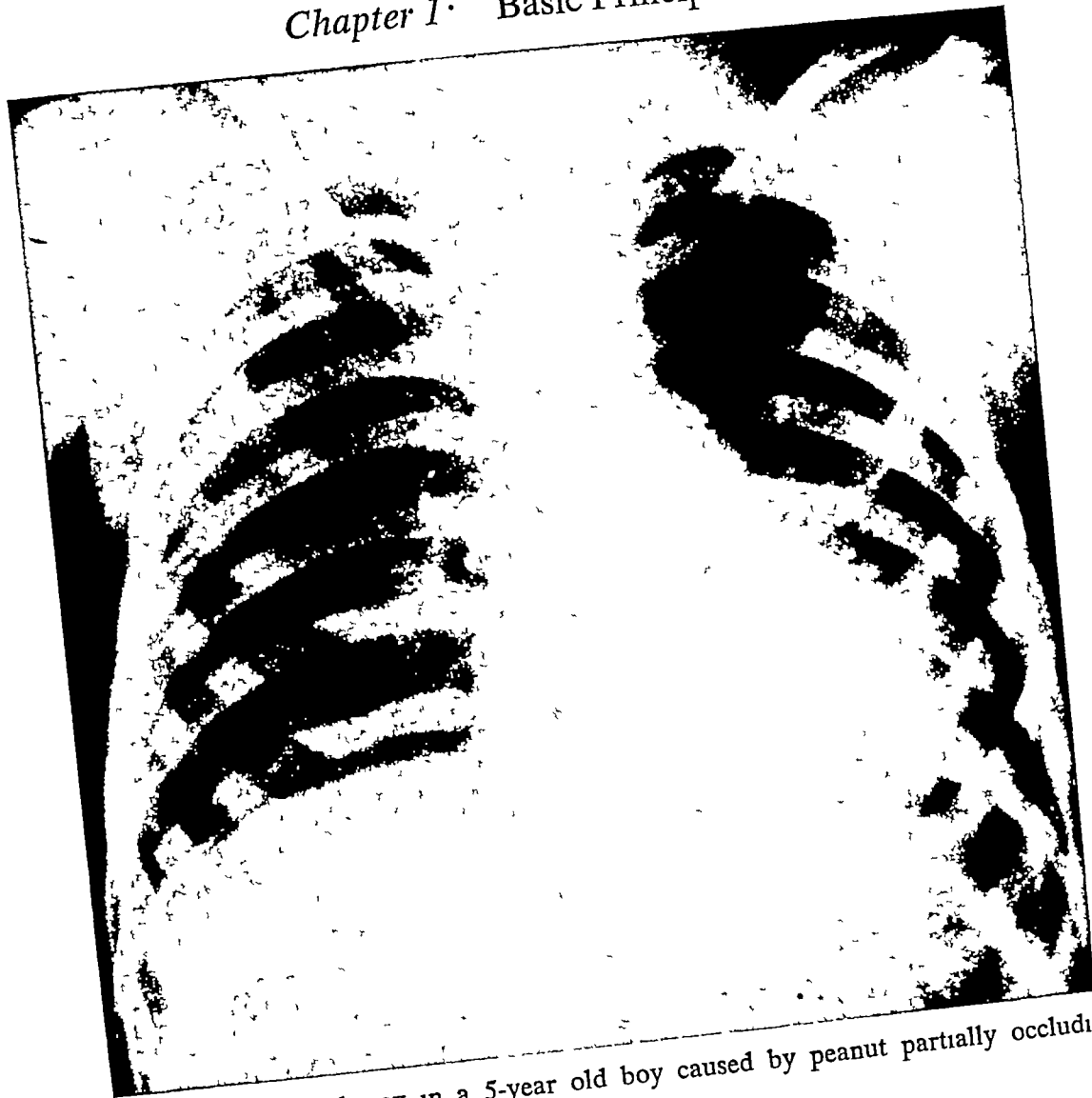


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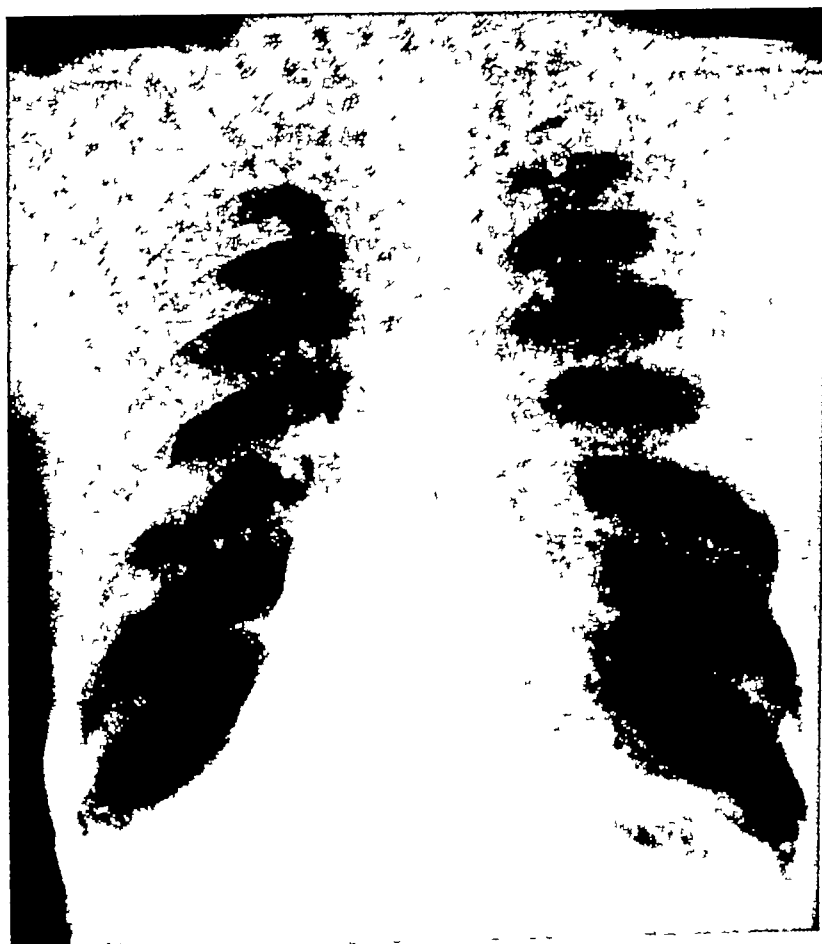


Fig 6C and D X-ray pattern of lobar atelectasis Right lateral view of middle lobar atelectasis and postero-anterior view of right lower lobar atelectasis



Fig. 6E and F X ray pattern of lobar atelectasis. Left lateral view of left upper lobe and postero-anterior view of atelectasis of left lung.

### PHYSIOLOGY OF THE PLEURAL CAVITY

The higher center controlling the remarkable reflex of respiration lies in the hind brain. The *mechanical act* depends on the interaction of five components:

- 1 An unimpeded airway,
- 2 An intact chest wall,
3. An intact diaphragm,
- 4 Atmospheric pressure,
- 5 The elastic recoil of the lungs

Functionally, the chest wall is a firm cage which protects the thoracic viscera not only from trauma but also from the constant atmospheric pressure of approximately 15 pounds per square inch.

**Normal Intrapleural Pressure.** By contracting and descending, the diaphragm increases the intrathoracic volume and negative pressure, with the result that air enters the lungs, and the inspiratory phase of respiration occurs. As air enters the pulmonary alveoli, the pulmonary elastic tissue is stretched. When the muscles of respiration cease to act, expiration—a purely passive act—follows from muscular relaxation and the elastic recoil of the lungs.

This interplay of factors creates a normal intrapleural pressure of  $-10$  cm of water on inspiration and  $-5$  cm of water on expiration. Because of free communication with the outside air, the pressure within the lungs is about one atmosphere, but the elastic recoil of the lung prevents this intrapulmonary pressure from being fully transmitted to the pleural cavity. The more the lungs are distended by inspiration, the lower becomes the intrapleural pressure, and the more the venous return to the right side of the heart is enhanced. If any one of these five components is altered, the change is immediately reflected in changed intrapleural mechanics and pressures.

**Increase of Negative Pleural Pressure.** This commonly occurs when a lobe or lung becomes atelectatic. As already mentioned, such atelectasis is followed by a relative emphysema of the remaining lung tissue, a shift of the trachea and mediastinum to the affected side, together with elevation of that leaf of the diaphragm and an approximation of the overlying ribs.

**Increase of Positive Pleural Pressure.** This follows a pneumothorax—be it spontaneous, artificial or tension—pleural effusion, hemothorax, or empyema. The increased pressure affects both the ipsilateral and contralateral lungs, the effect depending on the intrapleural pressure exerted.

When the positive pressure increase is small, the underlying lung falls away by elastic recoil towards the hilum, the so-called *relaxation collapse*. When, however, the intrapleural pressure rises above atmospheric pressure, the underlying lung is compressed by positive pressure—*compression collapse*.

Further, the heart, trachea, and mediastinum are forced to the opposite side, where in turn they compress the contralateral lung and reduce its ventilatory capacity. If the positive pressure rises above 15 to 20 cm of water, there is the additional hazard of obstruction of the venous return to the heart, and acute respiratory embarrassment.

The air from a tension pneumothorax may permeate

- (a) Directly through the parietal and visceral pleura into the soft tissue of the chest wall and mediastinum, or
- (b) Proximally in the peribronchial tissues to reach the mediastinum



Fig. 7 Residual fibrin on the visceral pleura of the left lung, preventing its re-expansion after liquefying, aspirating and draining a left hemothorax by water seal drainage.

When fluid compresses the lungs fibrin is deposited on the pleural surfaces thus further complicating the pleural lesion to be treated. While aspiration or simple drainage is often followed by total re-expansion of the collapsed lung, in those cases in which much fibrin has become deposited on the visceral pleural surface the fibrin may effectively hold the lung captive and permanently reduce its ventilatory capacity. This process can occur with remarkable speed, as shown in Figure 7. In this case a postoperative intrapleural hematoma though liquified by varidase left an effective fibrin vice on the underlying lung within three days of the original left lower lobectomy. From a practical point of view the problems of pneumothorax are also the problems of bronchopleural fistula.

**Bronchopleural Fistula.** When an emphysematous bulla, lung abscess, hydatid cyst or tuberculous cavity ruptures into the pleural cavity or when the lung is damaged by fractured ribs or by surgical resection air escapes into the pleural cavity through bronchopleural fistulas. The lung collapses as previously described.

A fistula is either *open* and in free communication with atmospheric pressure via the lung or *valvular* allowing air to pass into the pleural cavity but not out again, thus producing a tension pneumothorax. Pathologically open fistulas are usually large and valvular ones small. Clinically, bronchopleural fistulas with tension pneumothorax are distinguished by detecting tracheal deviation and cardiac displacement and are confirmed by measuring the intrapleural pressure with a pneumothorax apparatus. An "open" pneumothorax has an inspiratory pressure of  $-4$  cm. of water and an expiratory pressure of  $+4$  cm. of water. A tension pneumothorax may have an inspiratory pressure of  $+4$  cm. of water and an expiratory pressure of  $+12$  cm. of water.



- (b) Arteriovenous aneurysm, which is a shunt,
- (c) Obstruction of the pulmonary artery by emboli or obliterative arteritis

Naturally, these categories frequently overlap, as many lesions cause combined forms of insufficiency. The net result is *anoxia*

**Anoxia.** Following prolonged exposure to severe oxygen lack, very formidable after effects occur, for "*anoxia not only stops the machine, but wrecks the machinery*" (Haldane 9, 10). *Three minutes* of complete anoxia is enough, for in that time the brain is irrevocably damaged. But, as Haldane further emphasizes, *even partial anoxia*, or *hypoxia*, means not a mere slowing down of life but progressive and perhaps irreparable damage to living structures

Few practitioners can escape the problems of anoxia. Thus, the anesthetist may be faced with spasm of the vocal cords, or may have to deal with anoxia if he is slow to intubate after giving muscle relaxants. Anoxia faces the thoracic surgeon who is dealing with a "flail" chest wall, hemothorax, a blocked bronchus, or cardiac arrest. It applies equally to the chest physician with the problems of debilitating pneumonia, to the cardiologist with acute pulmonary edema, to the endocrinologist with diabetic coma, to the neurologist with bulbar poliomyelitis, to the neurosurgeon with comatose patients, to dental and orthopedic surgeons with jaw and cervical spine fractures, and—last but not least—to the general surgeon with his problems of postoperative pneumonia and atelectasis following abdominal operations

**TYPES OF ANOXIA** Physiologically, Samson Wright (11) distinguishes four types of anoxia

1. *Anoxic type* the tension of oxygen in the arterial blood is lower than normal, and consequently the hemoglobin is not saturated with oxygen to a normal extent,
2. *Anemic type* the oxygen tension in the arterial blood is normal, but the quantity of functioning hemoglobin is too small;
3. *Stagnant type* the arterial blood is normal in oxygen tension and oxygen content, but the oxygen is supplied to the tissues in insufficient amounts,
4. *Histotoxic type* the tissue cells are poisoned so that they are unable to make effective use of the oxygen supplied to them

Clinically, *hypoxia* developing over several hours gives a typical picture of cerebral dysfunction, the patient failing to concentrate, wandering in his mind, becoming restless, irrational or delirious, and having a cold, clammy forehead, rising respiratory and pulse rates, and falling blood pressure

Even shallow breathing, especially after thoracic operations or from pain or loss of lung substance, will cause hypoxia and must be prevented by oxygen, minimal sedation, and supervised deep breathing with the help of a physiotherapist.

*Anoxia*, when *acute* as from bronchial occlusion, gives sudden, transient tachycardia followed by a slowing and weakening of the pulse with deepening cyanosis.

**MANAGEMENT** Anoxia is far more easily prevented than treated, for, if of any duration, there is no lasting recovery. There are five essentials to check in any patient with anoxia:

1. Is there a firm, functioning chest wall and underlying lung?
2. Is the airway clear?

## References

- 3 Is there a supply of oxygen at 6 liters per minute?
- 4 Is the blood volume adequate?
- 5 Is the heart pumping?

*Seconds mean life* we must check quickly!

The importance of a firm chest wall and lung and of a clear airway have already been stressed in considering paradoxical respiration bronchopleural fistula and atelectasis. Treatment consists of adequate support to the chest wall, intercostal drainage to relieve pneumothorax or massive effusion and bronchoscopy to clear the bronchial tree (see Chapter 4)

*Oxygen Therapy* The most effective and comfortable method of giving oxygen is by using an oronasal plastic mask to which oxygen is delivered at the rate of 6 liters per minute. If this proves unsatisfactory the use of an oxygen tent is advised. The practitioner should never forget mouth-to-mouth insufflation, or a mask and a hand bellows. These are means of oxygen supply that make use of the 20 per cent oxygen in the air in which we live.

*Blood Loss* Hemoglobin is necessary for the transport of oxygen from the lungs to the tissues for oxidation. When blood loss is the cause of anoxia the patient is pale, sweating, wide eyed, blue lipped, and anxious. After difficult lung resections, an intrathoracic hematoma may cause this type of anoxia and may require replacement of the lost blood by transfusion and evacuation of the hematoma to restore lung function.

*A Beating Heart* The heart is a pump that normally expels blood at a pressure of 120 mm. Hg. If the pump fails as from sudden anoxia, it is as important to compress the heart manually by cardiac massage at pressures and rates equal to normal heart action as it is to clear the airway by bronchoscopy and to inflate the lungs with oxygen. Adams (12) restored normal rhythm from ventricular fibrillation after 1 hour and 50 minutes of manual compression to maintain an effective cerebral circulation. Prompt action and patient persistence can reward the surgeon who is aware of these basic facts. In Chapter 24 of this text, there is a further discussion of Cardiac Arrest.

## REFERENCES

- 1 Lister J. On the antiseptic principles in the practice of surgery. *Lancet*, 2: 353, 1867.
- 2 Johnson, J., and Kirby C. K. *Surgery of the Chest*, 1st ed., Chicago, The Year Book Publishers, Inc., 1952.
- 3 Sweet, R. H. *Thoracic Surgery*, 1st ed., Philadelphia, W. B. Saunders Co., 1950.
- 4 Brock, R. C. *The Anatomy of the Bronchial Tree*, 2nd ed., New York, Oxford University Press, 1954.
- 5 Report by the Thoracic Society. The Nomenclature of Bronchopulmonary Anatomy. *Thorax*, 5: 222, 1950.
- 6 Boyden, E. A. *Segmental Anatomy of the Lungs. A study of the pattern of the segmental bronchi and related pulmonary vessels*, 1st ed., New York, McGraw Hill Book Co., Inc., 1955.
- 7 Birnbaum, G. L. *Anatomy of the Bronchovascular System*, 1st ed., Chicago, The Year Book Publishers, Inc., 1954.
- 8 Birath, G., and Crafoord, C. Function tests in pulmonary surgery. *J. Thoracic Surg.* 22: 414, 1951.
- 9 Haldane, J. S. Symptoms, causes, and prevention of anoxaemia and the value of oxygen in its treatment. *Brit. Med. J.*, 2: 65, 1919.
- 11 Wright, Samson. *Applied Physiology*, 7th ed., New York, Oxford University Press, 1940.
- 12 Adams, R. In discussion, "Induced cardiac arrest for intracardiac surgical procedures," *J. Thoracic Surg.*, 30: 620, 1955.

## 2

### BASIC PROCEDURES IN THORACIC MANAGEMENT

Before considering any specific disorder, seven basic procedures common to the management of many thoracic lesions will be described. They are

- ✓1 Chest aspiration,
- ✓2 Intercostal water-seal drainage,
- ✓3 Continuous pleural suction,
- ✓4 Removal of an intercostal tube,
- ✓5 Changing an intercostal tube,
- ✓6 Bronchoscopic aspiration,
- ✓7 Tracheotomy

#### CHEST ASPIRATION

**General.** Chest aspiration may be either diagnostic or therapeutic.

*Diagnostic aspiration* will disclose if a pleural lesion such as a pneumothorax is under tension, or if pleural fluid is blood, pus, or clear effusion.

*Therapeutic aspiration* aims either at correcting disordered pleural mechanisms by removing air or fluid, or at other times enhancing therapy by introducing air, drugs, or antibiotics into the pleural cavity.

*Roentgenograms* Whenever possible, posteroanterior and lateral chest x-ray films should be studied before aspirating, and the site for aspiration should be chosen in relation to the fluid or air levels. In practice, there are few thoracic emergencies that are not further clarified by chest roentgenograms. Most emergencies other than acute anoxia from sputum retention or tension pneumothorax will allow time for roentgenography. In difficult cases, a fluoroscopic examination and marking of the site to aspirate with a skin pencil can be of tremendous assistance.

*Site for Aspiration* If the practitioner is familiar with the anatomy, thoracentesis can be performed through any intercostal space. The most common site is in the eighth or ninth intercostal space below the angle of the scapula. When an empyema loculates to the side or in front, aspirate over it. If there is an apical pocket of air, it is approached either from the front through the first intercostal space below the clavicle or from above through the posterior end of the first or second intercostal space.

*Needles* These must be checked to see that they are sharp, not too fine, and not blocked.

**Technic.** The patient is suitably sedated.

The key to successful aspiration is the use of a three-way locking tap that keeps the circuit intact and prevents unmeasured air from entering the pleural cavity.

*Position for Aspiration.* This depends on the site of the fluid.

(a) To aspirate in the eighth left intercostal space posteriorly, the patient sits up, folding his arms on a pillow or on a bed table. One pillow is placed flat against the buttocks as a support. When covered with a sterile towel, this pillow makes a satisfactory "table."

(b) To aspirate in the second left intercostal space in front, the patient rests comfortably on pillows with his head turned to the right.

(c) To aspirate the apex of the pleural cavity the patient sits forward, resting his arms on his knees or on a bed table

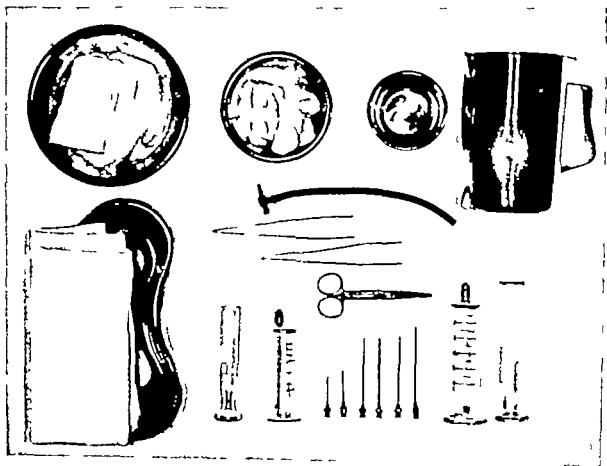


Fig. 1 Chest aspiration tray

CHEST ASPIRATION TRAY (FIG. 1)

*Sterile Equipment*

- 1 large tray
- 2 large kidney dishes
- 2 small bowls for antiseptic solution, swabs and chest dressings
- 2-pint measure
- dressing sheets, large and small

*Syringes*

- 1 20 ml. syringe with hypodermic needles, size 23-21
- 1 50 ml. syringe with 2 aspiration needles, size 17-15
- 1 3-way adapter and length of small rubber tubing

*Instruments*

- 1 scissors
- 1 artery forceps
- 2 dissecting forceps

*Lotions etc*

- antiseptic solution
- procaine solution 1 per cent, 20 ml.
- collodion
- specimen bottle for pleural fluid

*Unsterile Equipment*

- laundered mask and gown
- elastoplast roll
- scissors

**DIAGNOSTIC ASPIRATION** The skin over the area selected for aspiration is widely painted with antiseptic solution, and sterile towels placed in position. After checking the strength of the local anesthetic, preferably 1 per cent procaine solution, 10 ml are drawn into a syringe, and an intradermal wheal is raised with a fine needle. The needle is changed, and the underlying muscle and intercostal space are gently infil-

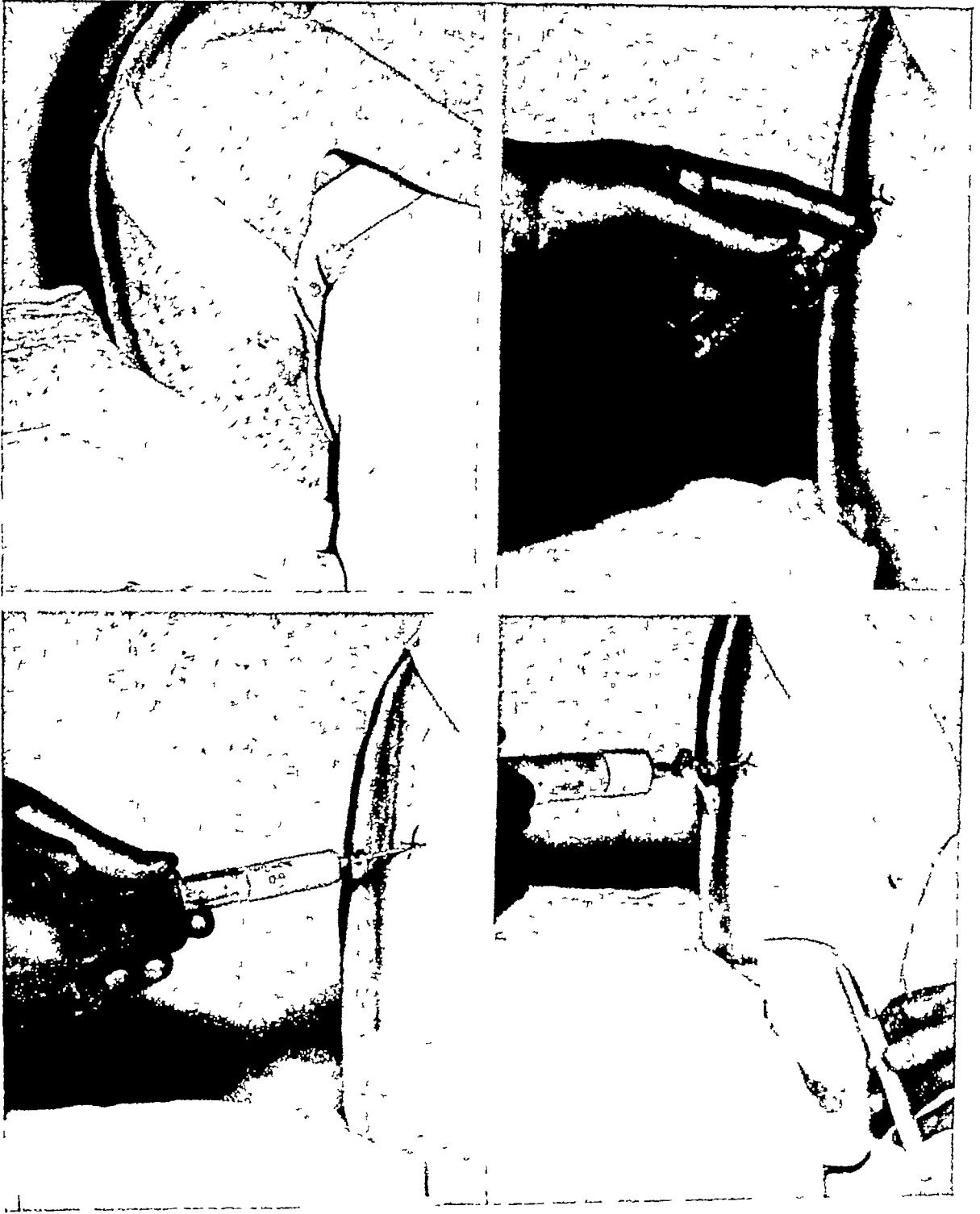


Fig 2A Technic of chest aspiration

*Upper left* Patient leaning forward with pillow support in front and behind X marks the spot chosen to aspirate

*Upper right* Infiltrating the intercostal space with local anesthetic

*Lower left* Diagnostic aspiration of pus

*Lower right* Therapeutic aspiration continued, using a 3-way adapter A specimen is being collected

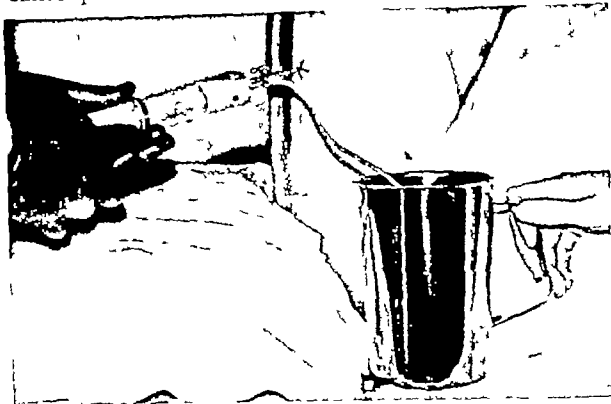


Fig. 2B Technic of chest aspiration continued.

trated. The needle should be introduced at the bottom of the space in order to avoid injuring the intercostal vessels for spiking of an intercostal vessel has been known to lead to an unexpected intrapleural hematoma. After entering the pleural cavity a specimen is withdrawn (Fig. 2). The findings are as follows:

- ✓ When air is withdrawn it may indicate
  - (a) A leaking syringe barrel or needle connection
  - (b) Puncturing the lung with the needle
  - (c) Air from a spontaneous pneumothorax or pyopneumothorax.
- ✓ When blood is withdrawn it may appear
  - (a) As a steady flow from an intercostal vessel
  - (b) Hemothorax
  - (c) From the lung, when it is usually frothy
  - (d) From the liver when fragments of liver substance may be detected if the aspirate is evacuated onto a swab
- ✓ When pus is withdrawn its origin is
  - ✓(a) An empyema,
  - ✓(b) A lung abscess, or
  - ✓(c) A subphrenic abscess

When pus is difficult to locate, repeated puncture may be necessary and even further fluoroscopic check and marking of the site may be required before aspirating.

**THERAPEUTIC ASPIRATION** The one essential for this procedure is that the aspirating apparatus be leakproof. If after diagnostic aspiration, further aspiration is required, a larger needle of size 15 to 20 caliber is firmly locked to a three way tap and syringe and is inserted along the line of local anesthesia. Syringes and fittings

with the B-D Luer locking connections are most serviceable for this purpose. Patients are still occasionally admitted to thoracic surgical units in acute respiratory distress because failure to observe this fundamental rule when aspirating an empyema thoracis has caused tension pyopneumothorax. For large aspirations, a 50 ml syringe is excellent, but for most a 20 ml size will suffice. When the aspiration is finished, the needle is removed and the skin puncture covered with a collodion pledget.

When the aspirate has a high fibrin content, the syringe may "seize up" during the aspiration procedure. It should then be removed, washed through with saline, and oiled with sterile glycerin or paraffin before continuing. Sterile petrolatum jelly, applied to the joints between needle, tap, and syringe, has the advantage of sealing any small leaks.

*After care* 1 Specimens of aspirate should be sent for

- ✓(a) Bacteriologic examination for organisms both aerobic and anaerobic,
- ✓(b) Cytology,
- ✓(c) Protein estimation and sugar content

✓2 The state of the lungs, pleural cavity, and mediastinum is thereafter checked by further roentgenograms, and any remaining faults are attended to.

### INTERCOSTAL WATER-SEAL DRAINAGE

Intercostal water-seal drainage is one of the simplest and most effective of thoracic operations. Its value in treating bronchopleural fistula was discussed in Chapter 1. The method was first introduced by Kenyon (1) in 1911.

Before inserting the drainage tube, however, it is advisable to determine the required point of entry by careful inspection of posteroanterior and lateral chest films.

#### TRAY FOR INITIAL INSERTION OF INTERCOSTAL DRAINAGE TUBE (FIG 3)

##### *Sterile Equipment*

1 large tray  
1 large kidney dish  
3 small bowls for antiseptic solution and sterile lubricant  
1 10-ml syringe  
swabs and chest dressing  
dressing sheets, large and small

##### *Needles*

hypodermic needles, No. 23 and No. 21  
aspiration needles, No. 17  
curved cutting needle and nylon suture

##### *Instruments*

1 scissors  
1 artery forceps  
2 dissecting forceps  
1 scalpel blade and handle  
1 trocar and cannula  
Malecot catheter introducer  
Malecot catheters, or straight rubber tubes  
1 needle holder  
sterile drainage bottle, rubber stopper with glass tubing,  
length of rubber tubing and glass connection

##### *Lotions*

antiseptic solution

procaine solution 2 per cent, 20 ml.  
sterile lubricant

## Unsterile Equipment

laundered mask and gown  
elastoplast for covering dressing, 1 roll  
scissors  
safety pins

**Technic.** The positioning of the patient and the method of infiltrating the chest wall with local anesthetic are the same as for chest aspiration. The steps are as follows:

- 1 On entering the pleural cavity withdraw the plunger of the syringe to confirm the presence of air pus, or blood.
- 2 Select a Malecot catheter which when stretched on an introducer, will pass through the cannula provided.
- 3 Check to see that the assisting nurse has the drainage tube with wide-bore glass connection ready and the bottle filled with sufficient water to create a seal. Check further to see that the drainage tube is *in fact* attached to the longer of the two glass tubes in the stopper of the bottle and that this glass tube reaches to the bottom of the bottle.
- 4 Nick the skin with a sharp scalpel and insert the trocar and cannula into the pleural cavity (Fig. 4). During this procedure the nurse supports the patient from the opposite side. Withdraw the trocar and place the thumb over the end of the cannula, then carefully insert the catheter stretched on its introducer.

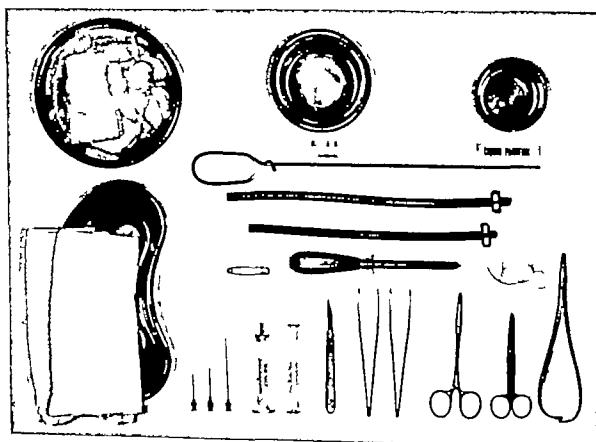


Fig. 3 Tray for insertion of intercostal drainage tube.



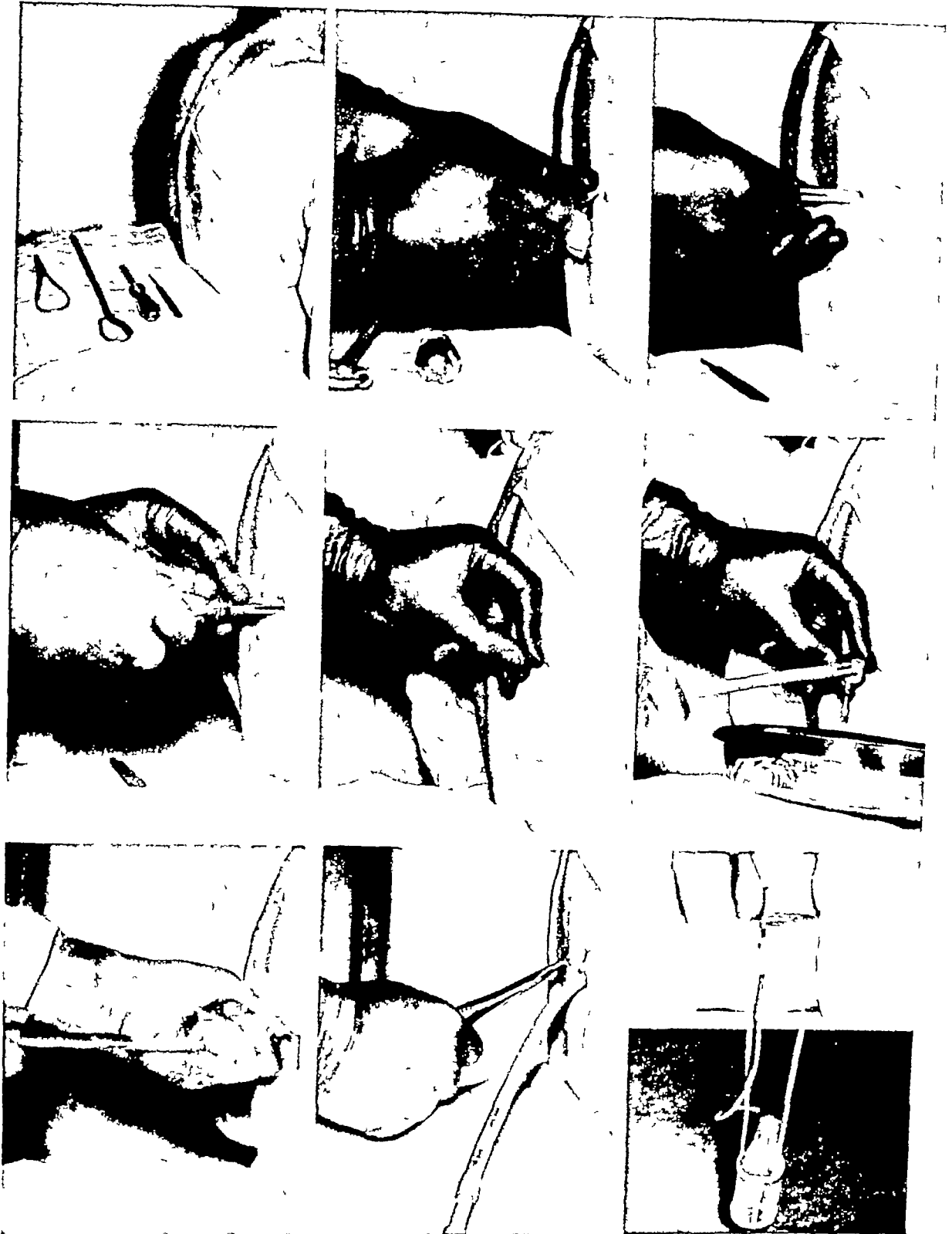


Fig 4 Technic of inserting Malecot catheter

*Top row* (a) The patient positioned and ready Local anesthetic has been injected (b) The skin is incised (c) The trocar and cannula are inserted

*Middle row* (a) The trocar is withdrawn (b) Left thumb held over the cannula (c) The Malecot catheter is inserted, and the introducer withdrawn

*Bottom row* (a) The cannula is withdrawn (b) Skin suture is inserted and the ends tied around the catheter (c) The tube is anchored to the patient's side with elastoplast and to the bed, leaving a length for the patient to be free to move There is a straight drop to the water-seal which hangs from the bed in a metal bottle holder

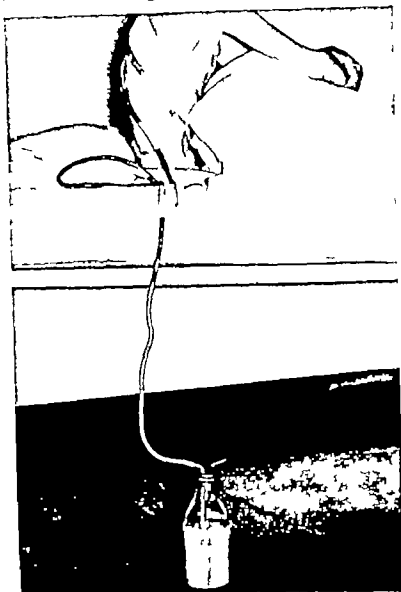


Fig. 5 Fixation of intercostal drainage tube. (Detail of Fig. 4)

An 8-inch length of elastoplast is placed above the tube and one below. A further length anchors the tube to the patient's side. A fourth length is placed around the tube and is pinned to the bed.

remove cannula and introducer and connect the catheter to the water-seal drain.

- 5 Secure the catheter with a skin stitch and further safeguard it with four short pieces of elastoplast—one above and one below the tube holding the dressing in place—one fixing the tube to the patient's side and one fastening it to the bed (Fig. 5)
- 6 With the patient once more in bed, check to see that the water-seal drain is functioning normally and that the fluid level in the glass draining tube swings normally with each breath

**After-Care of Water-Seal Drainage.** For effective drainage, not only should the tube bore be the same throughout (preferable  $\frac{1}{4}$  inch in diameter) but the rubber used should be thick enough not to be collapsed by suction. The system comprises the intercostal tube, a glass connection, and a long rubber drainage tube reaching down to the drainage bottle. At the bottle, the rubber tubing is attached to a length of glass tubing that reaches below the surface of water to form the water seal. A second short glass tube through the stopper allows air to escape

✓ With the increased negative pressure of each inspiration, the water rises up the tube; on expiration, the water level falls. In the presence of a bronchopleural fistula, with each cough air escapes from the bottle. Therefore, if fluid drains down and the tube oscillates with each breath, the system is in working order.

The following points, however, require constant checking

- ✓1. At no time must the bottle be raised above the level of the chest, lest fluid be aspirated into the pleural space and thus infect a sterile cavity or cause disaster with a bronchopleural fistula.
- ✓2. When the bottle is changed, the rubber intercostal tube must first be clamped to prevent air from entering the pleural cavity. Further, the bottle must be checked to see that the rubber drainage tube has been properly reconnected to the long glass tube.
- ✗3. The rubber drainage tube may become blocked
  - (a) If the patient sits on it,
  - (b) If it sags and the bend becomes filled with fluid,
  - (c) If fibrin clot forms at the glass connection, which is the narrowest segment;
  - (d) If the chest end of the intercostal tube becomes blocked by fibrin or by the expanding lung

These hazards can be prevented by ensuring that the rubber tube has no undue length, by regularly "milking" it down, and by changing the drainage tube daily.

- ✓4. If there is free leakage of air when using continuous suction and water-seal drainage, the leakage may come from one of three sources
  - (a) A leaking tube or an ill-fitting stopper,
  - (b) Air leaking in around the catheter, especially in empyema cases,
  - (c) A bronchopleural fistula

### CONTINUOUS PLEURAL SUCTION

The purpose of intercostal drainage is to allow re-expansion of the underlying collapsed lung, removal of pleural fluid, and, when present, closure of a bronchopleural fistula.

In certain circumstances—especially after lobectomy or following induction of an artificial pleurisy for spontaneous pneumothorax—when prompt re-expansion of the remaining lung tissue is desired, *continuous pleural suction* is necessary.

It is essential that the suction pump used be capable of shifting the minute volume of air leaking into the pleural cavity. The suction apparatus available varies with the hospital, and may be operated either from a portable high pressure electric machine or from a central "master" apparatus.

The author is not convinced that very high pressure is essential or even desirable. It is uncomfortable for the patient. Further, it is no more efficient than 5 cm Hg, and the drainage tube blocks far more rapidly, often before all fistulas have sealed themselves. *The one essential is that the visceral and parietal pleura be brought into apposition, by removing the volume of air that escapes from the lung, thus sealing any fistulas.*

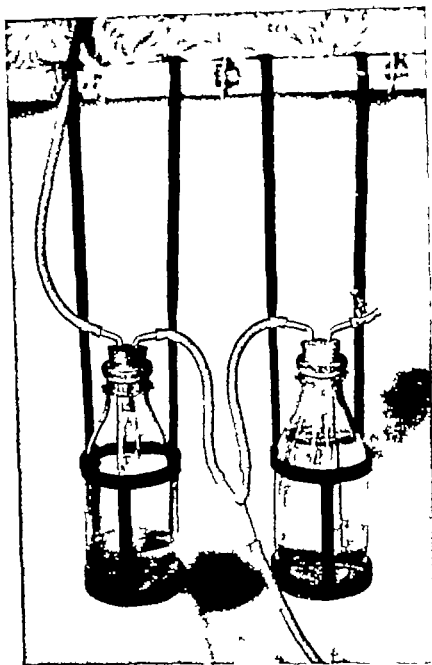


Fig. 6. Regulation of intercostal suction, with side bottle.

This simple device is useful where no pressure-reducing valves are available. By means of a Y connection, a second bottle is introduced into the tube system from the water seal drain to the wall suction. This second bottle is filled with water the long glass tube left free, and the rubber tube connected to the short glass tube. The effective suction pressure on the water-seal is then the difference in height of the two fluid levels.

A great disadvantage of continuous suction attached directly to the water-seal drain is variation in suction pressures. These variations can be reduced by inserting a side bottle with a glass T or Y connection to the circuit between pump and water-seal bottle. The side bottle is filled with water and connected to the system by a short tube. The long glass tube is left open to the air. Therefore, when the pump is in action, the effective pressure is the difference in levels between the side and main drainage bottles. If this is not sufficient, it may be delicately regulated by a screw clip on the side tube (Fig 6). Hospital engineers can also adapt pressure reducing valves for this purpose with the added advantage that the pressure used can be accurately measured on the valve gauge.

**Care of Tubes and Bottles.** The following points are important.

1. The tube must be firmly fixed into the patient. A method of doing this has already been discussed.
2. When the bed is being changed, the tube must be refixed.
3. The apparatus must then be checked to see that the fluid level can still swing freely.
4. The bottle must be changed daily, or more frequently if it fills in a shorter time. During the change of bottle, the tube is securely clamped so as to prevent any inrush of air into the pleural cavity.
5. When the tube has become blocked (usually in 24 hours), a roentgenogram is taken. If the lung is satisfactorily re-expanded, the tube is removed. If not, it is changed and suction continued.
6. The original level of water in the bottle should be marked with a strip of adhesive.

### REMOVAL OF AN INTERCOSTAL TUBE

This is one of the most common procedures the thoracic resident is called upon to do.

#### EQUIPMENT TRAY (FIG 7)

##### *Sterile Equipment*

1 large tray  
1 large kidney dish  
1 10 ml syringe  
2 small bowls for antiseptic solution  
dressing sheets, large and small  
swabs and chest dressings

##### *Needles*

hypodermic needles No. 23 and No. 21  
curved cutting needle and nylon suture

##### *Instruments*

2 dissecting forceps  
scissors  
1 needle holder

##### *Lotions*

antiseptic solution  
procaine solution 2 per cent, 20 ml

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##### *Unsterile Equipment*

elastoplast for covering dressing, 1 roll  
scissors  
laundered mask and gown  
bucket to receive soiled dressings and tube

## Removal of an Intercostal Tube

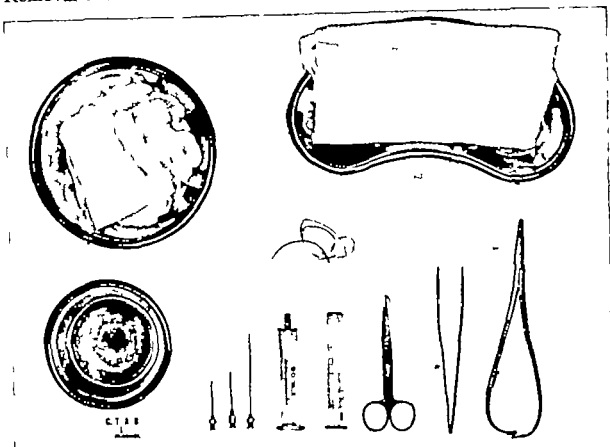


Fig. 7 Tray for removal of intercostal drain.

**Technic.** The removal of the intercostal tube is a sterile procedure which may be done with or without local anesthesia

**PROCEDURE WITHOUT LOCAL ANESTHESIA** This is satisfactory when a straight tube has been used. The dressing is lifted, the chest wall painted with antiseptic skin lotion, and the retaining suture cut with sterile scissors. The skin around the stab wound is held firmly between thumb and index finger, and the tube is carefully removed. A skin suture is inserted and a dressing applied. Alternatively a suture may be placed ready—but not tied—when the tube is originally inserted.

If there is evidence of sepsis around the tube no suture is used but the opening is covered with a moist antiseptic dressing and strapped firmly in place to prevent air from entering the pleural cavity.

**PROCEDURE WITH LOCAL ANESTHESIA** (Fig. 8) The method is the same as above except that a local anesthetic is first infiltrated around the area. This is of value with apprehensive patients or when Malecot catheters have been used.



Fig 8 Technic of removing intercostal drainage tube

*Upper left* The patient leans forward over pillows, and dressings are removed

*Upper right* Local anesthetic agent is injected

*Lower left* With the left thumb and index finger holding the area, the tube is pulled out

*Lower right* A suture is inserted without allowing entry of air into the pleural cavity

### CHANGING AN INTERCOSTAL TUBE

There are two occasions when this is required:

- 1 Changing a recently inserted, but blocked, intercostal tube,
- 2 Changing a short empyema tube.

1. When a Malecot catheter or plain tube has been used to drain a pleural space but has become blocked before the lung has completely re-expanded, the tube must be changed. If the tube has but recently been inserted and is in a satisfactory intercostal space, the tube is removed under local anesthesia, a Malecot catheter is stretched on an introducer, and a fresh tube is reinserted as previously described. If, as sometimes happens, the track is lost, it is readily reestablished with a trocar and cannula. If the first tube site is unsatisfactory, the tube is removed and a new tube is inserted in a fresh site.

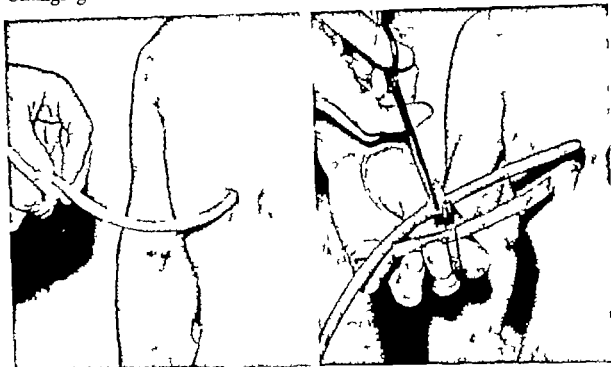


Fig. 9 Technic for changing short empyema tube.

*Upper left* After removing the old tube and dressings, a Jaques rubber catheter is inserted to gauge the exact length of the sinus.

*Upper right* The tube for re-insertion is made  $\frac{3}{4}$  of an inch shorter than the sinus length.

*Lower right.* The new tube is fixed with a pin and adhesive plaster strips, more being added if required.



2 When there is a short tube draining an intercostal space and sinograms have shown that the cavity is closing, the tube is removed, the length of the sinus track checked with an Allison empyema sound or a soft Jaques rubber catheter and a new tube inserted one inch short of the total length. The new tube is secured with a pin strapped to the chest wall and covered with a dressing (Fig 9)



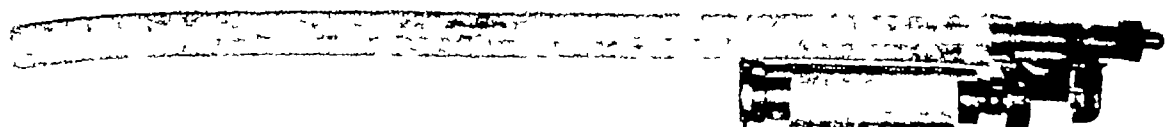


Fig 11A Method of keeping bronchoscope ready and sterile for emergency use  
After bronchoscope and suction tube are sterilized by boiling, they are kept in sterile plastic sheath made of plastic tube as shown

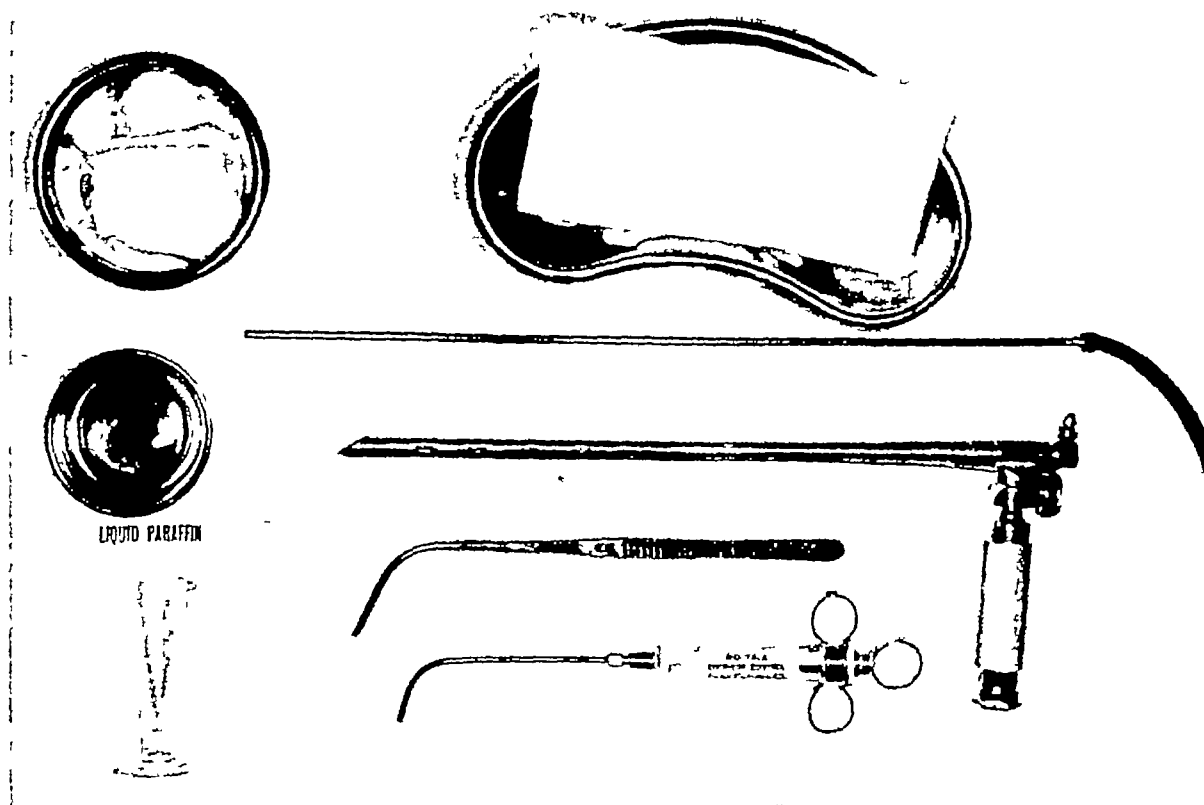


Fig 11B Tray for emergency bronchoscopy

A tray containing the following equipment is kept sterile in the operating theater

#### EQUIPMENT TRAY (FIG 11)

- portable bronchoscope, preferably of the Longworth pattern with battery attached
- metal bronchoscopic and rubber suction tubes
- Krause's laryngeal forceps
- laryngeal syringe
- measuring glass and pipet for local anesthetic
- 1 bowl with wool swabs for anesthetizing the throat
- kidney dish for waste
- 2 sterile dressing towels

#### Make sure that

- (a) The rubber suction tube fits the metal bronchoscopic suction tube,
- (b) Bronchoscopic light and suction tube fit,
- (c) The light is efficient

#### ADDITIONAL EQUIPMENT

- bottle of local anesthetic solution, e.g. lignocaine 2%
- laundered mask and gown



Fig. 12. Technic of local anesthesia for bronchoscopy. Left: Throat is gently painted, including epiglottis. Right: When the curved cannula is sited over the glottis, the patient inhales and the plunger is driven home.

**Preoperative Preparation.** The patient is placed in a room in which strong suction is available. If the head of the bed is not detachable, a stool is placed for the operator to stand on at the head end of the bed. The patient should be sitting comfortably and should be supported by pillows.

When bronchoscopy is urgently required for a semiconscious patient, no premedication is necessary. In less urgent circumstances, various drugs are given preoperatively although the patient takes nothing by mouth. Atropine sulfate gr  $\frac{1}{4}_{100}$  is given to stop salivation. A sedative (e.g. Omnipon gr  $\frac{1}{6}$  to  $\frac{1}{2}$  or scopolamine gr  $\frac{1}{8}_{50}$ ) may be given to allay apprehension. These drugs may conveniently be given intravenously. For a child, atropine sulfate gr  $\frac{1}{8}_{50}$  with Nepenthe (1 min. for each year of the patient's age) will suffice.

As the aim is to preserve and encourage the cough reflex, local anesthesia should be used except with children under six who are rarely cooperative and for whom a general anesthetic is to be preferred.

The patient is given an amethocaine lozenge (50 mg.) to suck on until it dissolves—within 10 to 15 minutes—by which time the mouth, tongue and throat will be numb. This is usually enough, but its efficacy should be tested by painting the throat with a 2 per cent lignocaine solution. Since some patients have an idiosyncrasy to local anesthesia, no more than 3 ml. should be used. The swab is mounted in Krause's laryngeal forceps, dipped in the solution, and gently applied to each vallecula and the epiglottis. When intratracheal anesthesia is also desired, the syringe is filled with 2 ml. of the solution and passed through the mouth so as to lie above the glottis. The patient is instructed to breathe out, and as he inhales again, the plunger of the syringe is driven home. He immediately coughs and scatters the anesthetic material throughout the tracheobronchial tree. The anesthetist's left hand should hold the patient's tongue with a gauze swab while his right hand sites the syringe with the fingers and drives home the plunger with the palm (Fig. 12).

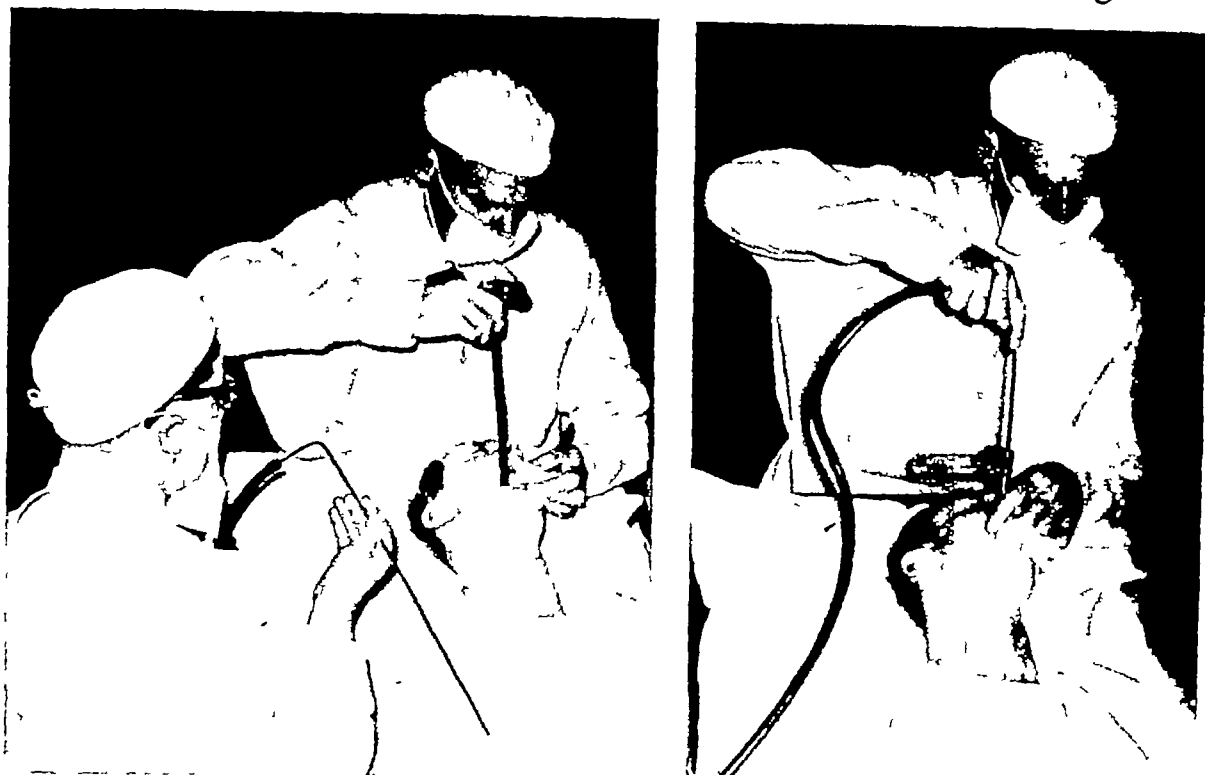


Fig 13 Technic for emergency postoperative aspiration bronchoscopy Left inserting the bronchoscope Right aspirating retained secretion

**Technic of Bronchoscopic Aspiration.** The patient sits up in bed with his back and neck supported on pillows and the head free for hyperextension. If the top of the bed is removable, the approach is easy. If the top of the bed is not detachable, the operator stands on a stool at the head end of the bed and leans over the patient.

The points to observe are (Fig 13)

- ✓ 1 Protect the patient's eyes with a towel
- ✓ 2 Check the light and the size of the bronchoscope, and lubricate the instrument with glycerin. A bronchoscope 7 mm in diameter suffices for adults
- ✓ 3 Check the suction apparatus, and see that there is at least six feet of suction tubing
- ✓ 4 Holding the bronchoscope in the right hand, gently draw the patient's tongue forward with the left hand and let him extend his head and elevate his chin
- ✓ 5 Pass the bronchoscope to the back of the pharynx and behind the epiglottis which is lifted forward to disclose the vocal cords immediately below. With the bronchoscope at first turned to the left so that the bevel is fore and aft, it is carefully passed between the cords
- ✓ 6 If no endotracheal anesthetic has been injected, the patient immediately coughs. The secretion, which usually lies in the trachea and at the origin of the main bronchi, is aspirated. Thereafter, a milliliter of local anesthetic may be injected. In turn, the left and right main bronchi are aspirated, and all secondary bronchial orifices are inspected
- ✓ 7 Even the most apprehensive patient is by this time relieved of his distressing tightness in the chest and is only too ready to assist by coughing and clearing the secondary bronchi. Further sputum, as it accumulates, is removed, and the secondary bronchi are cleared with a fine gum-elastic tight suction tube

The operator must be dextrous and he should not prolong the procedure unduly. If the patient is cyanosed, oxygen is given via the side tube of the bronchoscope.

Finally as the instrument is withdrawn the trachea is again inspected and any residual secretion sucked clear.

**Postoperative Management.** The following points are important

- ✓ 1 The patient is allowed to have nothing by mouth for two hours after bronchoscopy that is, until the effects of all local anesthesia have worn off and the pharyngeal reflex has returned to normal.
- 2 The effectiveness of the bronchoscopy is confirmed by clinical and radiologic examination.
- ✓ 3 If re aeration is not complete further physiotherapy and postural drainage are necessary.
- ✓ 4 The lung is checked by daily roentgenograms until aeration is maintained.
- ✓ 5 If the patient cannot cooperate and repeated bronchoscopy is required, the efficacy of this treatment must be reassessed and the advantages of tracheotomy must be fully considered (see page 40)

**Complications.** There are five complications of bronchoscopy

*Preoperative*

- ✓ Reaction to local anesthetic.

*Operative*

- ✓ Hemorrhage
- ✓ Rupture of the bronchus

*Postoperative*

- ✓ 4 Laryngeal spasm
- ✓ 5 Laryngeal edema

①

**PREOPERATIVE COMPLICATION** *Reaction to Local Anesthetic* This is a very rare complication it is preventable and, with care in the quantities of anesthetic used should never occur. However if a reaction does occur the operator must have a clear plan of action. The reaction is easily recognized for the patient keels over and develops a paroxysm of generalized muscular spasm.

✓ An endotracheal tube must be passed at once with the aid of a laryngoscope the cuff inflated, and the spasm overcome with Pentothal and a relaxant injected intravenously.

2 The patient is then artificially respired with a hand bellows or anesthetic machine until the drug has been detoxified. In one such experience this took over half an hour and required two injections of relaxant.

3 If the endoscopist has none of the above-cited equipment and drugs at hand he must immediately pass the bronchoscope through the tightly closed glottis and respire the patient by mouth to bronchoscope insufflation until an anesthetist can be summoned to assist—usually a matter of about 10 minutes.

✓ *To leave a patient in unrelieved spasm for over two minutes is usually fatal*

When the collapse is accompanied by cardiac arrest as well, an epigastric incision and opening of the diaphragm allows the surgeon to perform cardiac compression. The nurse in attendance should at the same time do artificial respiration until an anesthetist can relieve her.



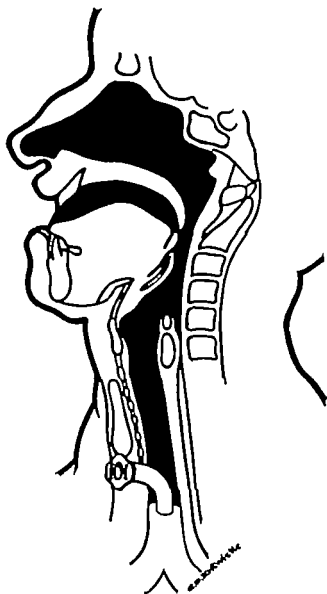


Fig. 14 Physiologic advantages of tracheotomy dead space is bypassed resistance to breathing is reduced.

Carter and Giuseffi (3) point out that, beside these mechanical advantages tracheotomy has equally important physiologic functions (a) it decreases the amount of dead space in the respiratory tree with a resulting increase in effective ventilation and (b) it decreases resistance to breathing, during both inspiration and expiration (Fig 14) As a corollary in crushing injuries to the chest there is diminished excursion of the loose segment of the chest wall, and relief of pain (4) For these reasons too the recent use of tracheotomy in postoperative respiratory difficulty is firmly established. Its value in the case of debilitated, pneumonic, cardiac, or comatose patients has yet to be more widely appreciated.

**Equipment.** "In cases of dire necessity the operation has been undertaken successfully with a pen knife in such circumstances the addition of a hair pin to act as a retractor may be considered a luxury" (5) Sterile sets of equipment, however are usually available

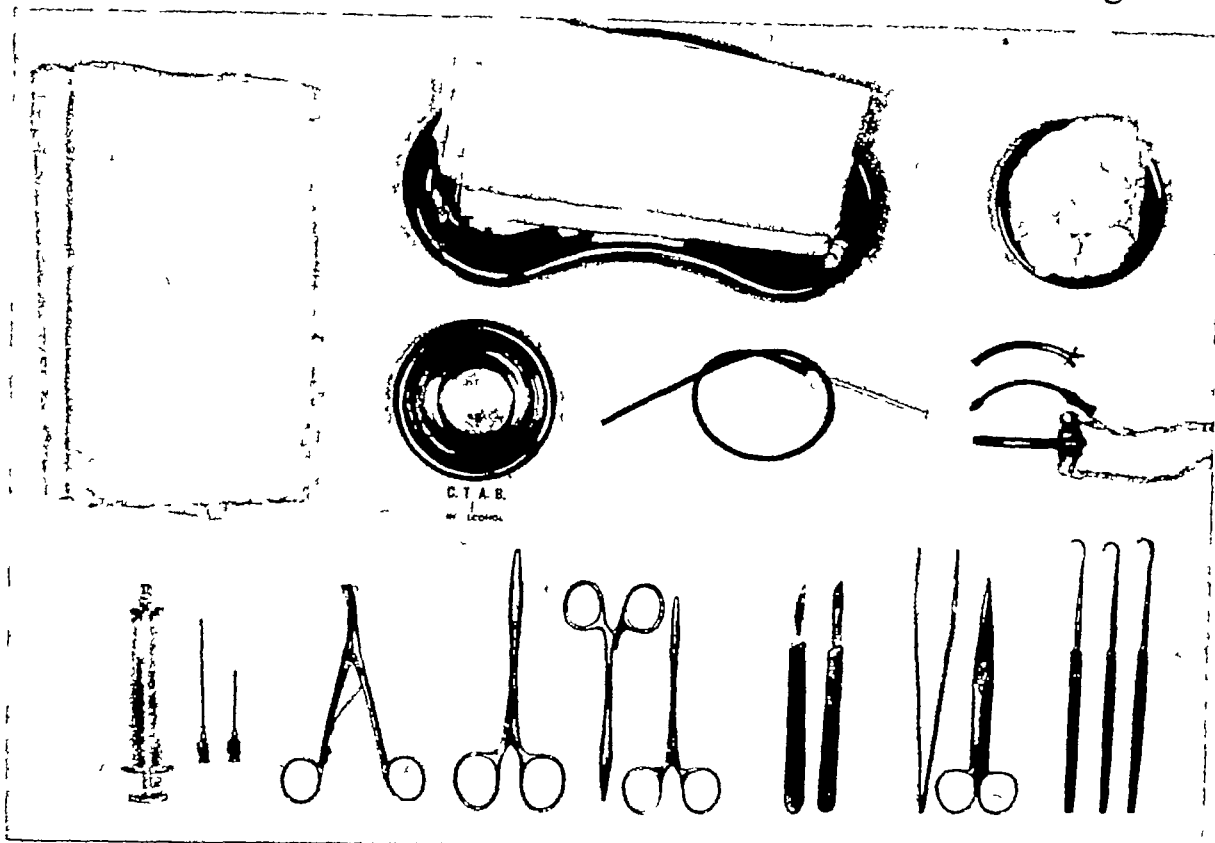


Fig 15 Tracheotomy set (the essentials)

## TRACHEOTOMY SET (FIG 15)

*Sterile Equipment*

- 1 large tray
- 1 kidney dish
- 2 small bowls for antiseptic solution and swabs
- 2 trolley covers to cover table and patient's bed
- 4 dressing sheets to drape wound
- 1 square of lint for leaving behind the tracheotomy tube
- cotton and gauze swabs

*Syringe and Needles*

- 1 5-ml syringe
- local anesthetic needles No 22 and 24
- curved cutting needles No 12 and 16
- nylon suture, 00

*Instruments*

- 2 Bard-Parker handles, No 3 and No 4, and blades
- 1 fine dissecting scissors
- 1 suture scissors
- 1 toothed dissecting forceps
- 1 nontoothed dissecting forceps
- 1 tracheal dilator
- 2 double lock retractors
- 2 single lock retractors
- 2 Dunhill artery forceps
- 2 mosquito forceps
- graduated set of tracheotomy tubes 1 to 18
- fine rubber catheter with glass connection
- file of catgut ligatures

*Lotions*

- antiseptic solution
- procaine solution 1 per cent, 20 ml

## EMERGENCY HIGH TRACHEOTOMY

**Technic.** If only for his own protection, especially in the presence of diphtheria or pulmonary tuberculosis, the operator must wear a mask and gown

It is a tremendous asset to have an anesthetist giving oxygen and holding the patient's head centrally and extended over a towel pillow or sandbag. The larynx and trachea thereby become prominent and palpable. A suction apparatus must be in readiness. The steps in the operation are as follows

- 1 Infiltrate the skin with 5 cm. of 1 per cent procaine solution.
- 2 Make a vertical midline incision  $1\frac{1}{2}$  to 2 inches downward from the cricoid cartilage dividing the platysma (also the anterior jugular vein if it crosses the operative field) and the sternohyoid muscles. If time permits, clamp the bleeding points
- 3 Palpate the trachea and rings with the left index finger. draw the thyroid isthmus down, and expose the upper three tracheal rings. Clamp and divide the isthmus if necessary
- 4 Holding the scalpel short with the fingers insert it into the trachea, cut upward for half an inch (i.e. 2 or 3 rings) and then rotate the blade to spread the tracheal incision.
- 5 Introduce the dilating forceps remove the scalpel and insert the tracheotomy tube with its obturator then remove dilator and obturator
- 6 Relieved of obstruction, the patient takes a deep inspiration and immediately coughs retained sputum into the wound. Suck the sputum clear and cleanse the trachea and bronchi by slipping a fine catheter down the tracheotomy tube whose wings are firmly held with the thumb and index finger against the neck.
- 7 Now tie the tube tapes securely around the neck, insert the inner tube and tie any skin sutures

*Points to remember are*

- 1 Unless the head is well extended, the trachea is difficult to locate and passes down and back into the chest.
- 2 Bleeding will cease under pressure alone once the obstruction is relieved
- 3 The action of the dilating forceps is exactly opposite to most other hinged surgical instruments. it is normally closed, and pressure causes it to open. This can be confusing when first encountered.
- 4 In the case of the child *in extremis* quickly pass a bronchoscope aspirate the bronchial tree inflate the lungs with your own breath and, when breathing has started again, complete the tracheotomy with the bronchoscope in position. This fixes the trachea and facilitates the operation
- 5 If respiration has failed, artificial respiration is required using the Sylvester method or tube and air bellows attached to the tracheotomy tube. If the heart action has ceased, cardiac compression is required forthwith

## LOW TRACHEOTOMY

This operation is usually performed when there is no urgency in definitive surgery of the trachea. The position is the same as for high tracheotomy; the incision



reaches from the cricoid cartilage to the infrasternal notch, and the muscles are retracted as before. The left innominate and inferior thyroid veins require care. The thyroid isthmus is retracted upward or divided, the trachea, lying deeply, is located, and a long tracheotomy tube is inserted.

### SHELDEN'S PERCUTANEOUS TRACHEOTOMY

This new technic employs an instrument consisting of three parts: 1, a hollow needle provided with a special slot and lateral opening to serve as a grooved director; 2, a set of cutting blades, one of which is tipped with a ball that can be passed through the lateral opening, advanced along the slot and passed into the pointed end of the needle; and 3, the tracheotomy tube which is thus guided into place and then used as a sheath through which the cutting blade is withdrawn. The technic is simple and rapid when used in adult patients without tracheal deformity or displacement (6).

**Postoperative Management.** Basic requirements include a well-ventilated, dust-free atmosphere of 70° F. and constant nursing attention, for a patient with a tracheotomy tube cannot call for help.

At the bedside should be a tray containing a duplicate tracheotomy tube, a tracheal dilator, artery forceps for turning the lock for the inner tube, swabs and dressings, a fine catheter, and suction apparatus or syringe.

The inner tube must be removed regularly (approximately every four hours), cleaned with water and a pipe cleaner, and boiled before it is reinserted.

Secretions are aspirated as soon as they are coughed up. The nurse must also be shown how to cleanse the bronchial tree by quickly inserting the fine catheter into the trachea and aspirating secretion.

*Infants* require special attention. An infant has such a small trachea that the closed end of the aspirating catheter can become a plunger which pushes secretions down into the bronchi, thus causing bronchial obstruction. For this reason, the closed end of the catheter is cut off for use on infant patients, and additional side holes are made in the catheter with a fine leather punch.

### TRACHEOTOMY IN INFANTS

The tendency for infants to cake endobronchial secretions after tracheotomy makes their after-care most exacting. Such infant patients require the strictest observation, for an infant's trachea is about the size of a lead pencil in diameter, and any caked secretions will quickly block the entire airway. Only emergency bronchoscopy through the tracheotomy opening and endoscopic removal with 3 mm peanut grasping forceps can save the day. In these circumstances, a special staff, prepared for emergency bronchoscopy no matter at what hour, can save these infant lives.

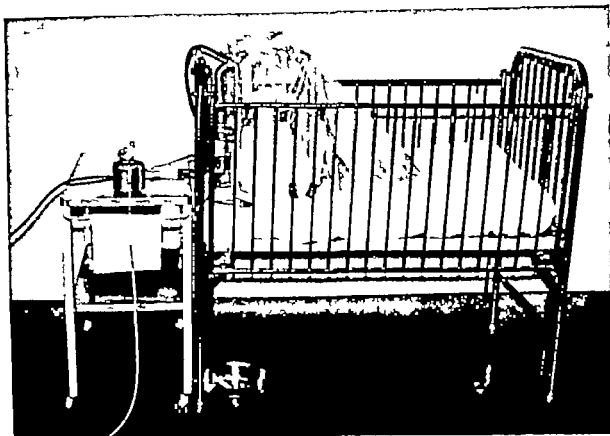


Fig. 16. "Croupette" fitted for "cold steam" and aerosol detergent.

The following points help prevent onset of this distressing complication

- 1 Place the baby in a croupette (Fig. 16) in which water is nebulized to produce cold steam. The inflowing air is cooled with ice water to 70° F
2. Lead in a second tube containing nebulized detergent such as alevaire (7). Experience has shown that even this dual attack of keeping the atmosphere both moist and impregnated with detergent may still not prevent caking of secretions
- 3 The author has found that instilling four drops of varidase solution down the tracheotomy tube every two hours arrests this unwanted process

*Removal of the Tube* Before the tube is removed, the patient should first have it half-corked, then completely corked, for 24 hours each. In infants paraldehyde sedation has proved effective.

In the author's experience infectious laryngeal edema of infants does not subside quickly requiring at least two weeks. Similarly if tracheotomy has been done following an operation on the lung, it is wise to leave the tube in place for approximately two weeks until the wound is healed and the patient's fear of pain with coughing has been abolished.

*At all times a spare sterile tracheotomy tube complete with all its parts must be ready beside the patient's bed for emergency changing if the one in use should become blocked.*

#### REFERENCES

- 1 Kenyon, J. H. A preliminary report of a method of treatment of empyema in young children, *M. Rec.*, 80 816, 1911

- 2 Gordon, W Perforation of the trachea and bronchus by the bronchoscope, *Thorax*, 5 369, 1950
- 3 Carter, B N , and Giuseffi, J Tracheotomy, a useful procedure in thoracic surgery, with particular reference to its employment in crushing injuries of the thorax, *J Thoracic Surg* , 21 495, 1951
- 4 Baronofsky, I D , Dickman, R W , and Venderhoof, E S The treatment of acute chest injuries, *Minnesota Med* , 33 49, 1950
- 5 Malley, C P In *Pye's Surgical Handicraft*, 16th ed , Bristol, John Wright & Sons, Ltd , 1949
- 6 Shelden, C H , Pudenz, R H , and Tichy, F Y Percutaneous tracheotomy, *J A M A* , 165 2068, 1957
- 7 Borrie, J , and Begg, N C Humidification in respiratory disorders with aerosol detergent, *New Zealand Med J* , 54 178, 1955

## PREOPERATIVE AND POSTOPERATIVE MANAGEMENT OF THORACOTOMY

Although any *preoperative* care of thoracic surgical emergencies is of necessity minimal, the principles involved are based on those used for nonurgent operations. Further, once the emergency has been corrected by thoracotomy, *postoperative* care is the same as for many nonurgent operations. It is therefore felt reasonable to review what thoracotomy involves before discussing thoracic emergencies in detail.

**Lesions Requiring Thoracotomy** Thoracotomy is performed for lesions of the

- |                           |                 |
|---------------------------|-----------------|
| 1 Chest wall              | 5 Esophagus     |
| 2 Pleural cavity          | 6 Mediastinum   |
| 3 Lungs                   | 7 Diaphragm     |
| 4 Heart and great vessels | 8 Upper abdomen |

Thoracotomy is comparable to laparotomy but, because of the open pneumothorax created by operation and its effect on the lungs, the postoperative care is more complex.

There are three standard *approaches* to the chest cavity:

- 1 The posterolateral by far the most common
- 2 The anterolateral,
- 3 The sternal

With the *posterolateral approach*, the scapula is mobilized by dividing the muscles attaching it to the chest wall and spine, i.e. the trapezius and the rhomboids above and the latissimus dorsi and serratus anterior below. With the *anterolateral approach*, the pectoralis major muscle is reflected and the serratus anterior and latissimus dorsi muscles partially divided. In both these approaches, the chest wall is traversed either by an intercostal incision or through the bed of the resected rib. The *transsternal approach* may require *vertical division* of the upper or lower half of the sternum or of its entire length. Alternatively, *transverse sternal division* and entering intercostal spaces on each side gives ready access to the anterior mediastinum.

As these various incisions of themselves interfere with postoperative arm, chest wall, and lung movement, the first aim of preoperative management is the safeguarding of existing arm and lung function.

### PREOPERATIVE MANAGEMENT OF THORACOTOMY

Preoperative management of thoracotomy includes

- 1 Physiotherapy
- 2 Assessment of lung function
- 3 Oral and nasal hygiene
- 4 Care of skin
- 5 Laboratory examination of sputum, urine, and blood,
- 6 Consideration of antibiotics and chemotherapy

- 7 Preoperative chest roentgenography,
- 8 Preoperative medication,
9. Intravenous therapy

**Physiotherapy.** This is of value to patients of all ages in gaining their confidence, encouraging them, improving their outlook, and, if possible, making them ambulant to improve leg circulation and lessen the chance of postoperative venous thrombosis. There is also opportunity for teaching correct posture, full range of arm movements, controlled diaphragmatic breathing, movement of local areas of the chest wall, and postural drainage. This supervision may extend up to the moment of operation, especially with bronchiectasis.

Nothing is more trying to the patient, physiotherapist, surgeon, or nursing staff than a convalescence hampered by inadequate preoperative physiotherapy. Yet, of necessity, this state must occur in emergencies and requires the understanding co-operation of all of the staff involved.

**Assessment of Lung Function.** In nonurgent cases, when thoracotomy is done for lesions other than pulmonary, lung function is usually assumed to be adequate. Its ventilatory sufficiency may be checked by measuring vital capacity and maximal breathing capacity.

When, however, because of disease of the lung, there is parenchymatous insufficiency—either quantitative from loss of lung parenchyma or qualitative from impaired diffusion of gases through the alveolar wall—or when there is any insufficiency of the pulmonary circulation from blockage or shunt, then special estimations of lung function by differential bronchspirometry are advised.

**Oral and Nasal Hygiene.** As septic teeth and mouths predispose to lung abscess formation or may aggravate postoperative lung complications, whenever possible thoracotomy should be delayed until all oral sepsis has been treated and the affected teeth either filled or removed. Similarly, when time permits, patients with infected nasal sinuses and tonsils should have these treated before thoracotomy.

**Care of the Skin of the Chest.** It is axiomatic that the skin of the patient's chest be healthy, yet occasionally young males are referred for operation with their backs covered with acne vulgaris. Such patients respond well to twice-daily hot baths, expression of comedos and pustules, washing the skin with soap and water or Cetavlon, and drying with alcohol and ether. Ultraviolet light has further improved the texture of such a skin. However, it may take some weeks to clear up these superficial infections.

Under normal circumstances, 12 hours before thoracotomy the skin of the neck, chest, and upper abdomen, both front and back, and of the axillas is prepared by shaving, washing, drying with alcohol and ether, painting with antiseptic solution, and covering with a sterile towel. The skin of the forearms is shaved in readiness for blood transfusion.

Finally, before the patient leaves the ward for the operating room, the skin is again painted with alcohol and antiseptic solution and left covered with a sterile towel strapped to the chest wall. In an emergency, there is usually only time for this latter preparation.

**Sputum Examination.** In nonacute chest investigations, sputum is examined by direct smear and culture for pathogenic organisms, and by smear, culture, and, if necessary, guinea pig inoculation for acid-fast bacilli. Five serial morning specimens of sputum are examined for acid-fast bacilli, and, when these are negative and a sus-

## Preoperative Management

picious lung shadow persists then 24-hour specimens of sputum are collected and concentrated. If these prove negative, laryngeal swabs or fasting gastric juice specimens are obtained. Bronchoscopic washings sometimes give the suspected positive sputum and the examination of sputum following bronchography may show a reluctant positive result.

It is surprising how the sputum examination can be forgotten and how tuberculous patients can be observed for weeks without changing a diagnosis of "solitary congenital cyst of the lung" because of omitting this investigation. Persistently negative outpatient sputa may become positive only when the patient is observed in hospital.

Sputum cytology has achieved new significance when stained by the Papanicolaou technic which is claimed to give 90 per cent positive results in carcinoma of the lung.

**Urine Examination.** Chronic lung sepsis not infrequently gives rise to nephrosis or amyloid disease. The former must be carefully noted and assessed before operation. Albuminuria must not be lightly overlooked.

*Diabetes mellitus* has proved no bar to successful thoracic operations, provided the disease has been stabilized and the insulin dose before and after operation properly adjusted by the attending physicians.

The state of the *prostate gland* must always be assessed and any obstruction removed, for it is as useless to treat hiatus hernia first in a man with benign prostatic hypertrophy and urinary obstruction as it would be to treat an inguinal hernia first. Postoperative urinary retention from benign prostatic hypertrophy has sometimes proved a troublesome complication even in thoracic surgery.

**Blood.** Patients with surgical chest complaints require a blood check on admission to hospital or when referred for surgical opinion. This includes red and white cell counts, hemoglobin estimation, complete blood grouping, erythrocyte sedimentation rate and, if indicated, differential white cell count. Where routine Wassermann determination has been the rule for years there has never been any reason to regret that routine. Occasionally the diagnosis of puzzling mediastinal and pulmonary shadows or sternal lumps has been established by a positive Wassermann or hydatid complement-fixation test.

Wherever there is a history of esophageal obstruction or prolonged sepsis blood urea and proteins should be estimated. If amyloid disease is suspected, gum biopsy and Congo red tests should be done. Patients who are to have continued postoperative intravenous therapy or those on low sodium intake require checking of sodium and potassium electrolyte levels before operation to provide a baseline for future therapy.

When the operating schedule is completed, the amount of blood required for each operation is estimated and blood specimens sent for crossmatching. The blood must be left in the blood bank and removed only as required.

**Antibiotics and Chemotherapy** During the period from 1945 to 1956, most thoracic operations were performed under cover of antibiotics. For adults, penicillin was usually given in doses of 500 000 units twice daily with streptomycin 1 gm. per day. These doses were scaled down according to age for children. The antibiotic injections were commenced a day before operation. However recent experience with resistant strains of various organisms, especially staphylococci, has served to emphasize that there is no substitute for the fundamental surgical principles of strict asepsis and hemostasis: that many single thoracotomies require no antibiotics.

that antibiotics are better reserved for prophylaxis when there is a specific risk of infection

**Preoperative Chest Roentgenogram.** Within 24 hours before operation, a chest film is taken and is kept on view in the operating room for inspection by both surgeon and anesthetist before anesthesia is commenced. When a foreign body is to be removed, the film should be taken on the way to the operating room.

It is surprising that changes in lesions are detected by last-minute films. In one patient believed to have a right-sided pericardial cyst, the preoperative film showed both colon and "cyst" filled with barium. It transpired that a resident had given the patient a surreptitious swallow of barium the day before. Overnight, the barium had passed into the transverse colon and now showed the lesion to be a right anterior colon-containing diaphragmatic hernia!

The rule requiring preoperative films is even more important when treating pulmonary tuberculosis which may have altered between the times of consultation and operation.

**Preoperative Medication.** This varies with the age of the patient, the type of anesthetic, and the operation. It is customary for anesthetists to see all chest surgical cases some time before operation and to order the preoperative medication themselves.

For children, rectal Avertin or Pentothal is desirable. Adolescents and adult females do well with Omnopon 11 mg and scopolamine 0.43 mg one hour before operation. For men, unless they are feeble, Omnopon 22 mg and scopolamine gr 1/150 (0.43 mg) is usually adequate.

**Venoclysis.** Finally, before the patient leaves the ward, venoclysis is commenced in the radial vein of the arm on the side opposite to the planned chest-wall incision. No. 16 to 18 needles are used in preference to cannulas, which destroy the veins. Alternatively, in children or in adults with short arms or poor veins, it is advisable to induce anesthesia first. Thereby the veins become more obvious and easier to enter.

### POSTOPERATIVE MANAGEMENT OF THORACOTOMY

Postoperative management is as important in an emergency as in a planned thoracotomy. During the operation, despite positive pressure anesthesia, the lung partially collapses, and the mediastinum is temporarily displaced to the opposite side. The aim of postoperative care is to restore the patient to normal as soon as possible with minimum discomfort.

The *problems* are to

- 1 Re-expand the lung and center the mediastinum by removing the pneumothorax,
- 2 Remove postoperative pleural effusion as it forms,
- 3 Prevent pleural and wound sepsis;
- 4 Prevent pulmonary complications;
- 5 Restore lung function, particularly ventilatory function,
- 6 Maintain nutrition;
- 7 Restore the patient to such useful employment as his condition permits.

**Removal of Pneumothorax.** As patients for thoracotomy are usually anesthetized with an endotracheal tube and positive pressure technique, air is effectively removed from the pleural cavity simply by fully inflating the lungs while the ribs are approximated and the intercostal muscle bundles sutured.

The air is as certainly removed by leaving an intercostal rubber tube attached to a water-seal drain. The tube should lie in the posterior axillary line at the level of the dome of the diaphragm.

It is important to see that the patient does not lie on his intercostal tube and obstruct it, for, in the presence of a large lung fistula, this invites tension pneumothorax, collapse of the lung, and rapidly spreading surgical emphysema.

*Immediately after thoracotomy* a chest roentgenogram is taken. If the lung is fully expanded and there is no lung fistula, the intercostal catheter can be removed then and there. Generally speaking it has been found preferable to leave it in position for 24 to 48 hours, as it also allows drainage of postoperative pleural effusions.

*Return to the Ward* In most thoracic surgical clinics it is customary for the anesthetist to supervise the return of the patient to the ward. Continuous oxygen is given as required. As the patient leaves the operating theater specific instructions are given to the ward nurse in attendance regarding

- 1 Blood transfusion and intravenous therapy
- 2 Oxygen therapy
- 3 Chemotherapy
- 4 Sedation
- 5 Tubes, bottles and suction pumps
- 6 Physiotherapy

*Blood Transfusion* If the blood loss has been replaced during operation, the blood drip is removed. However, if more blood or intravenous fluid is required, the drip is usually adjusted at the rate of 40 drops per minute. An accurate fluid exchange chart is kept and balanced at a specific time daily. To stimulate blood formation, each patient receives from therapy, e g. colliron, one dram thrice daily.

*Basic Observations* The pulse and respiratory rate and the blood pressure are recorded every 15 minutes, and any sudden or progressive change—such as a rise of pulse rate of 10 points or a fall in blood pressure below 100 mm Hg—is reported immediately. When the patient is fully conscious and his blood pressure has remained for three to four hours at 110 mm. Hg, he is allowed to sit upright.

*Sedatives* Sedatives such as omnopon or pethidine are given for relief of pain according to the patient's size and age.

*Removal of Pleural Fluid.* Serosanguinous pleural fluid collecting after thoracotomy comes partly from oozing at the site of operation and partly from pleural reaction to operative handling. The amount varies considerably; it may be insignificant or as much as 1 to 2 liters. The most fluid usually forms within the first 24 hours after operation and drains away through the intercostal catheter. This catheter rarely functions after 30 hours as its pleural opening becomes occluded by the lung, diaphragm and chest wall or its lumen blocked with fibrin. It is then removed.

If no catheter has been used, the amount of pleural effusion is watched clinically by serial roentgenograms and/or fluoroscopy. If at all significant in amount and not absorbed after three days it is aspirated, a specimen sent for culture and a further roentgenographic check made. Though formerly antibiotic solutions were left in the pleural cavity recent experience is against this practice. As a check of full lung re-expansion, the patient is finally x-rayed before leaving the hospital.

*Care of the Tube and Bottle* Every bottle used as a water seal should be of 2-liter capacity, have at least half a liter of sterile water in it, and have a glass tube leading one inch below the level of the fluid. The level of the fluid is marked before use so



that the amount draining each day can readily be estimated. Check to see that the rubber drain is connected to the glass tube that leads under the water and not to the shorter glass tube

When these bottles are to be sterilized by autoclaving, the rubber tube should be lightly wound around the bottle and not autoclaved while connected to the glass tubing, as the tube otherwise becomes permanently vulcanized, kinked, and blocked

When the catheter is patent, the fluid level in the glass tube swings up in response to the negative pressure of inspiration, and falls on expiration. If there is too little fluid in the bottle so that the lower end of the glass tube is exposed on inspiration, then the water seal is immediately broken, air enters the pleural cavity, and the lung collapses. This can easily occur in the wards when the bottle is changed daily

When changing the drainage tube and bottle, the intercostal catheter is clamped, the glass connection unhitched, and a freshly prepared sterile bottle connected before the clamp is released. As this procedure, apparently so simple, can yet be wrongly performed, even allowing air to enter the chest, it must be carried out or checked by the ward supervisor or by senior nurses trained in chest surgical procedures

#### **Removal of Intercostal Catheter.** (See Chapter 2 )

**Prevention of Pleural and Wound Sepsis.** Thoracic surgery has been made safe not only by improvements in anesthesia and in blood transfusion but also by better surgical technics and the use of antibiotics, which have greatly reduced the incidence of postoperative infections of the pleural cavity and wound

Strict sterile technic and absolute hemostasis are essential for uncomplicated healing. The intercostal and muscle layers must also be accurately and carefully apposed. Controversy still exists as to the relative merits of catgut, silk, or cotton suture material. This is largely a question of personal preference, but, if the operation has involved a septic focus, nonabsorbable sutures should be avoided as troublesome stitch abscesses may form later

The writer has found that a carefully placed subcutaneous layer of interrupted fine plain 00 catgut sutures, together with fine nylon skin sutures, allows suture removal within seven days. The subcutaneous layer also ensures careful apposition of the skin edges, excellent healing, and fine supple scars. If wound infection does occur, free drainage must immediately be established by releasing the sutures

**Chemotherapy** Preoperative chemotherapy is continued into the postoperative phase until the danger of sepsis has passed and the patient's temperature is normal, usually within one week. However, when sepsis is a complication, the antibiotic agent may be changed according to the sensitivity of the infecting organism. When there is no infection, chemotherapy is best avoided entirely

**Prevention of Pulmonary Complications.** Sputum retention is the most important pulmonary complication, leading to collapse of the lobe or lung, and, if untreated, to bronchopneumonia, abscess formation, or even death. All this train of events is preventable

Tenacious sputum from mild bronchitis may be loosened with Friar's balsam inhalations, and with expectorants such as the following, taken in hot water:

#### **MIST. AMMON. CARB**

Ammon carb	450 mg	Syrup tolu	2 ml
Tinct. camph co. min.	2 ml	Aq chlor to ½ oz.	15 ml

Nebulized water and detergents are also valuable

Every patient should have an anteroposterior film of his chest taken with a portable x ray machine on the day immediately following operation, and, if all is well, at intervals of not longer than three days for the first week. These films show the position of the mediastinum and the state of the lung and of the pleural cavity. The daily state of the lung is made known to the associated physiotherapists. The pleural problems have already been described.

If the lung or segment of lung is collapsed from retained sputum, vigorous postural drainage is commenced. When this fails to re-expand the lung, or when the patient is exhausted or unable to cooperate then postoperative bronchoscopy is required, as described in Chapter 2.

**Restoration of Ventilatory Function.** Ventilatory function depends on the bellows action of the diaphragm and chest wall, together with the ability of the lung to expand in response to the negative pressure created in the chest cage. Thoracotomy, by traversing the chest wall and causing pain with every breath and movement, interferes with this function and increases susceptibility to pulmonary complications. The discomfort after thoracotomy can be considerably lessened by intercostal nerve block with 5 ml. of a long-acting local anesthetic such as proctocaine.

*Physiotherapy* holds the key to success. The closest cooperation with a team of physiotherapists is essential for correcting postoperative posture, supervising and encouraging diaphragmatic breathing, coughing, and full arm, trunk, and leg movements, and for preventing pulmonary complications. The *exercises* taught in the pre-operative period should be repeated hourly and, during coughing, the chest must be firmly supported by physiotherapist, nurse or doctor. *Posture* is best corrected by the patient viewing himself in a large mirror wheeled to the foot of the bed. *Early ambulation* is encouraged but should not be hastened in the elderly who may develop cardiac arrhythmia, including auricular fibrillation, if forced to walk too soon. Due regard too must be paid to the *reason* for operation. For example if mitral valvotomy has been done for mitral stenosis with heart failure it is advisable to "make haste slowly." As the patient increases his own range of exercise, he will attend physical training classes in the massage department and will ultimately regain full chest movement and ventilatory function.

**Nutrition.** Few patients take more than a fluid diet during the first 24 hours after operation. On the second postoperative day light gastric diet can usually be given and quickly raised to a normal diet. As potassium loss also follows thoracic operations due amounts of potassium for replacement are required.

**Return of the Patient to Full Employment.** It is surprising how often in recent years when men have had chest operations and have apparently been restored to full health, they have not only failed to return to useful employment but have even registered as disabled persons. The question, "How serious are chest operations?" is asked by many patients, it requires an understanding reply. The time has long since passed when a man recovering from a chest operation can consider himself a freak. Occasionally even now a surgeon or a family doctor may unwittingly fail to encourage his patient back to full employment. That patient, though physically fit, may then remain a mental invalid. It behooves all who are concerned with such patients to see them regularly at follow up clinics, preferably one month after discharge from hospital again in three months again in six months and finally annually. A physiotherapist should be in attendance at the follow-up clinic so that any related problems can be discussed and arrangements made for their correction. In

addition, vital capacity is measured, breathing checked, and posture and movements inspected

Every encouragement should be given to the patient, especially by his family doctor, to return to a full, normal, useful life. For some elderly patients, return to work is not possible. Nevertheless, they should be encouraged to do all that they can for themselves instead of remaining chronic invalids. Such is possible only when correct mental attitudes can be maintained.

## Chest Wall Emergencies

### 4

## MANAGEMENT OF FRACTURED RIBS AND STERNUM

**Introduction.** Though fractures of the ribs are commonly regarded as trivial injuries the complications can be so serious and often clinically so obscure that all such cases should be treated with respect. As Watson Jones (1) aptly states "Children and adolescents seldom sustain fractures of the ribs but the risk of this injury increases with advancing years as the chest wall becomes more rigid. It should be recognized that the rarity of these fractures in children is the consequence of elasticity and flexibility of the chest wall, which obviously increases the danger of injury to the viscera within the chest. The degree of internal injury cannot be judged by the injury to the ribs. Similarly in adults, it is fallacious to assume that a trivial rib fracture cannot be associated with more than a trivial lung injury."

The injury varies in severity from a single uncomplicated fracture to the gross injury and multiple fractures of a modern automobile accident. *This chapter in the main refers to patients who have had less than three ribs fractured*

### PATHOLOGY

**Fractures of Ribs.** The most common cause of rib fractures in both war and peace is *external trauma* especially fore-and-aft blows to the chest wall. Rarely the ribs are fractured when the chest is crushed in a crowd. *Internal trauma* from violent coughing, straining, or heavy lifting has occasionally caused fractured ribs (2, 3, 4, 5) even in the later months of pregnancy (6).

As with all fractures, there are four possible types of rib fractures: *simple*, *compound*, *complicated*, and *pathologic*. The term "simple" is self-explanatory. *Compound* fractures arise either when the causative agent penetrates skin and pleura or when the rib ends are driven out through the skin. *Complicated* fractures have associated visceral injuries. *Pathologic* fractures arise on rib cysts or secondary neoplastic deposits.

The site of the fracture depends on the direction of the force and the convexity of the ribs and is most common near the costal angles. In the over all picture Hinton and Steiner (7) have emphasized that

- 1 The fifth to ninth ribs usually bear the brunt of trauma
- 2 Indirect trauma by approximating the rib ends increases their curvature so that they break outward near the middle of the shaft and, though such trauma usually involves more than one rib, it rarely produces complications
- 3 Direct trauma however produces fractures of one or more ribs at the point of impact, the traumatizing force thereby causing sharp rib fragments to penetrate the pleura and lung with complications.



Fig 1A Severe crush injury to left chest, with multiple fractures and deformity

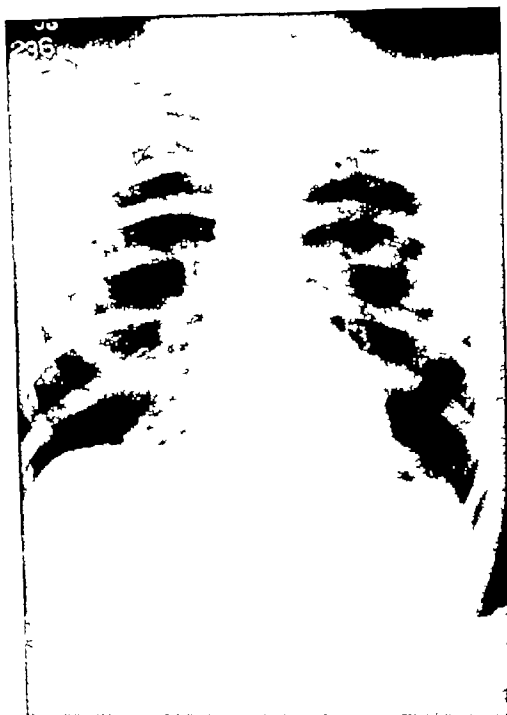


Fig. 1B Film three months later showing return of normal chest contour. No specific treatment had been given.



Fig 2A Fracture of ribs Simple fractures of left ribs three to eight

4. *Muscle violence* usually breaks the rib nearer the front and subperiostally, the seventh to eleventh ribs being most frequently involved
5. *Single* fractures are usually end to end, but multiple fractures may overlap

**Fractures of the Sternum.** The fracture line is usually transverse at or near the junction of the manubrium with the body of the sternum and is invariably associated either with rib fractures or costochondral or chondrosternal dislocations. The fracture may show (a) no obvious deformity, (b) a backward angulation with overriding of the fragments, or (c), if compound in type, a wound opening into one or both pleural cavities.

Holderman (8) emphasizes the statistical evidence that "simple fractures of the sternum are exceedingly rare." Regarding cause, he says that Gurlt (9) in 1864 reported the most common causes to be falling with the breast against a solid body, compression of the chest, falling from a height, a blow, thrust or step on the chest, and muscular action. Gurlt cited six instances of double fracture and two of triple fracture, all associated with other fractures. Nowadays, the sternum is most commonly fractured by a blow from a steering wheel or when the victim is struck by a vehicle (10).

The *prognosis* is good for uncomplicated injuries, but bad for the complicated. Gurlt reported 8 deaths in 54 uncomplicated lesions and 44 deaths in 45 complicated ones. Modern methods have naturally improved this picture, but the basic fact remains, "any complicated chest injury is serious."

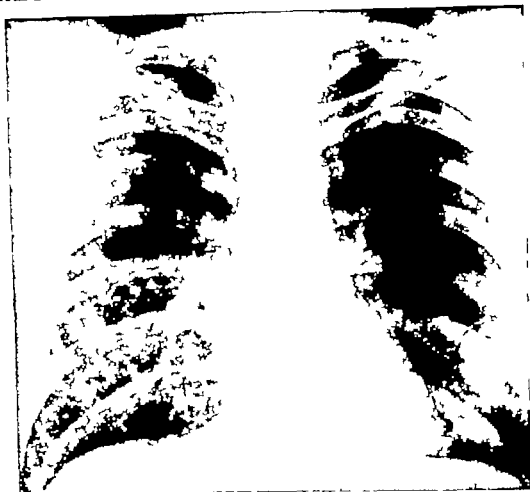


Fig 2B Fracture of ribs. Pathologic fracture right sixth rib

**Healing of the Fractures.** The intercostal muscles and surrounding ribs give considerable support to the fractured rib ends nonunion of simple fractures is unknown. When the underlying lung is functioning normally even the most markedly displaced fractures will unite in their normal position (Fig 1)

Callus forms within a fortnight, the fractures become stable and although roentgenographic signs of union may be delayed for some months functional recovery is complete in five to six weeks

### CLINICAL FEATURES

**Symptoms.** Pain with each breath or cough is the most common symptom and the pain can usually be well localized. With coexisting pneumothorax, there is a sense of tightness in the chest which is aggravated when tension pneumothorax displaces the mediastinal viscera to the normal side

**Physical Signs.** *General* Breathing is short and shallow. Steadily increasing surgical emphysema may also develop with pitting of the skin on pressure and a characteristic crackling sensation. Flail chest is recognized by the indrawing of the affected area with each inspiration and a return to normal contour on expiration. In the presence of multiple fractures the patient characteristically bends forward with his head toward the affected side in order to reduce movement. The fracture site is tender to the touch, and crepitus may be felt.

*Sternal Signs* Sternal fractures without displacement may not at first appear obvious, but pressure will elicit pain, crepitus, and possibly undue mobility. When there



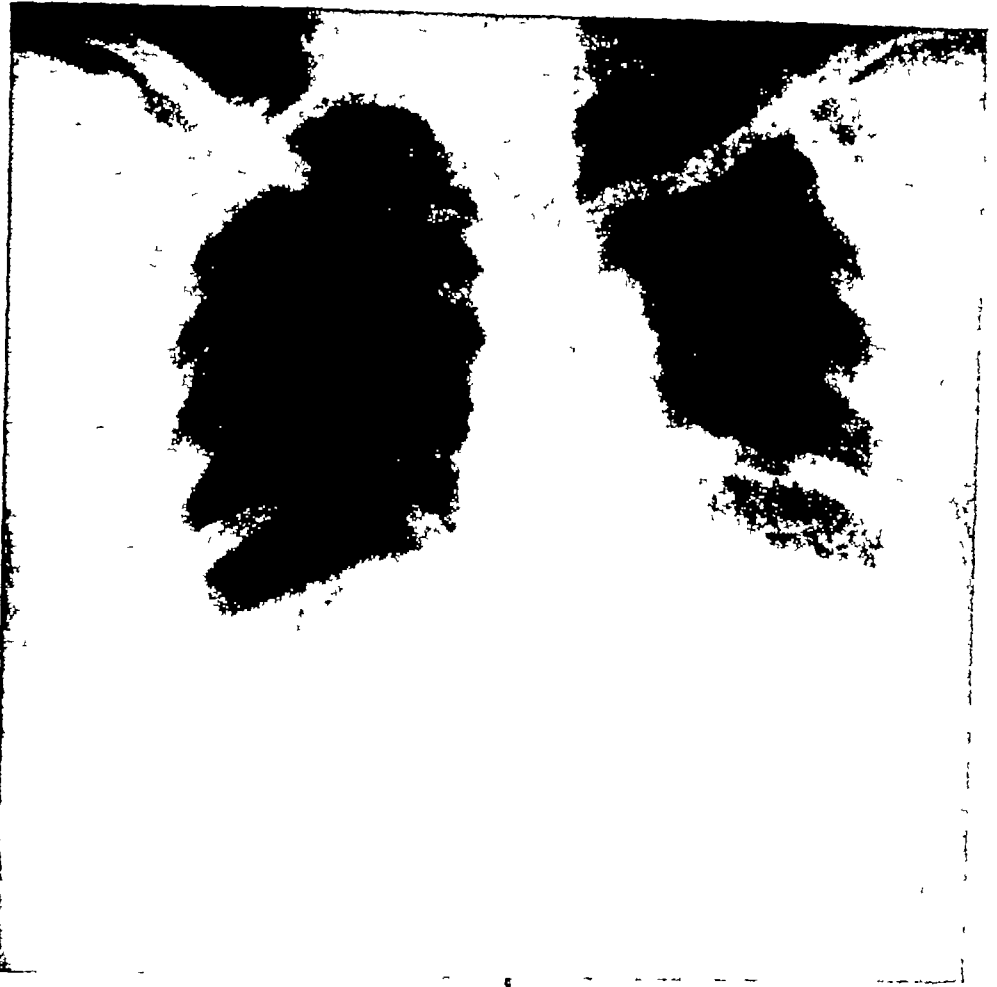


Fig 2C Fracture of ribs Complicated fracture with right pneumothorax

is displacement with either overriding or depressed sternum, the deformity is usually clearly visible

**Pleural Signs** Hyperresonance and diminished air entry confirm the presence of *pneumothorax*, while dullness to percussion, diminished air entry, and egophony confirm a *pleural effusion*. With *hemothorax*, signs of blood loss are added. These may be slow to develop and can be confirmed by diagnostic chest aspiration.

**Pulmonary Signs** These are related to the degree of lung damage. With minimal lung contusion, there is blood-stained sputum. Even with mild injury, fear of pleural pain may inhibit effective coughing and quickly lead to sputum retention, blocked bronchus, and atelectasis of a lobe or lung with corresponding physical signs. In the gross lung damage of major trauma, the patient may cough up pus and quickly show signs of relative anoxia, such as dusky color, incoordinated breathing, and a rapidly falling blood pressure.

**Cardiac Signs** With hemopericardium, there is cardiac tamponade, indicated by (a) muffled heart sounds, (b) falling blood pressure and (c) increased jugular venous pressure.

**Roentgenograms.** Chest films are essential to show the site and extent of fractures and the state of the pleural cavities, lungs, pericardial cavity, and mediastinum (Figs. 2A-D). Sternal fractures require lateral films for proper recognition. Massive emphysema is easily recognized by roentgenograms; lateral view shows retrosternal air. They can, however, be very deceptive, for they may not show fracture sites and certainly may not indicate the gravity of the chest injury.



Fig. 2D Fracture of ribs. Fracture of ribs complicated by sputum retention, atelectasis and mediastinal displacement to right.

repeated at half hour intervals or longer as the patient recovers from his injury, are of utmost value in assessing the progress of complications

**Diagnosis.** Diagnosis is usually obvious. It is suggested by the history, confirmed by physical examination, and accurately localized by chest roentgenograms

#### MANAGEMENT OF FRACTURED RIBS

*The principles of treatment are to*

- 1 Ensure adequate aeration of lungs
- 2 Relieve pain
- 3 Restore chest wall function
- 4 Anticipate and prevent such complications as atelectasis
- 5 Treat existing complications

#### CASES WITH MILD SYMPTOMS

The majority of patients are in this group

In young males as a first aid measure the pain of fractured ribs can usually be adequately relieved by elastoplast support. The chest is shaved, the patient breathes out, and 3 inch elastoplast is applied in overlapping strips, stretching from at least 2 inches on the sound side behind to 2 inches on the sound side in front. *Less than*

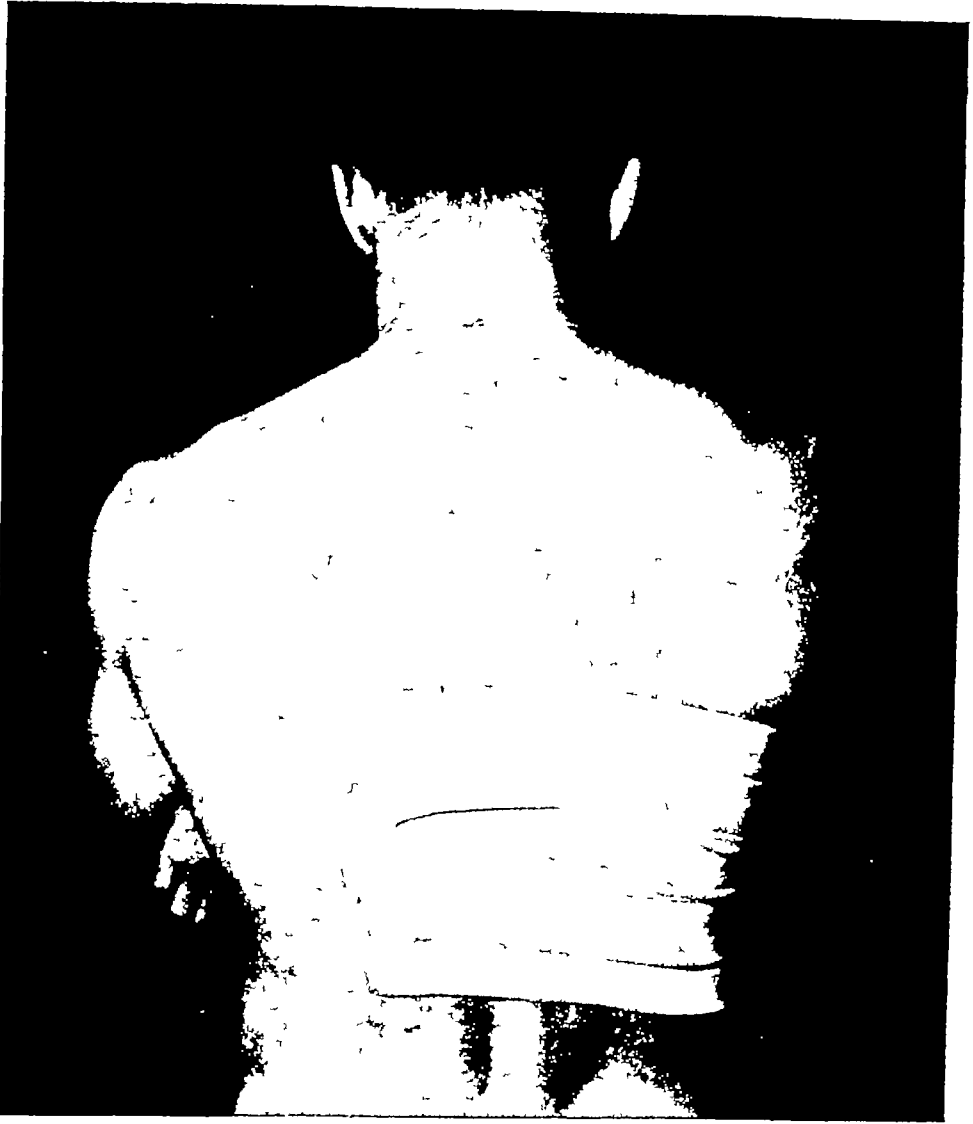


Fig 3 Method of strapping chest Elastoplast must cross midline in front by the same amount

*this is useless* If this support proves inadequate, encircling the chest wall with elastoplast fixes it and allows only diaphragmatic breathing (Fig 3) This support can usually be removed after two weeks

When the fracture is posterior and subscapular, the skin is abraded, or respiration is embarrassed by pain or pneumothorax, elastoplast support is useless

When the patient is immobilized from some other reason such as a fractured leg, bed rest alone may suffice for uncomplicated fractured ribs

#### CASES WITH SEVERE SYMPTOMS

When pain is constant, annoying, or severe—especially in middle-aged or elderly patients—it may be promptly relieved and the risks of sputum retention greatly lessened by *intercostal nerve block* This was first suggested by Latteri (11) who used alcohol Rovenstine and Byrd (12), and Harmon and others (13), reported marked and lasting relief of pain by procaine block

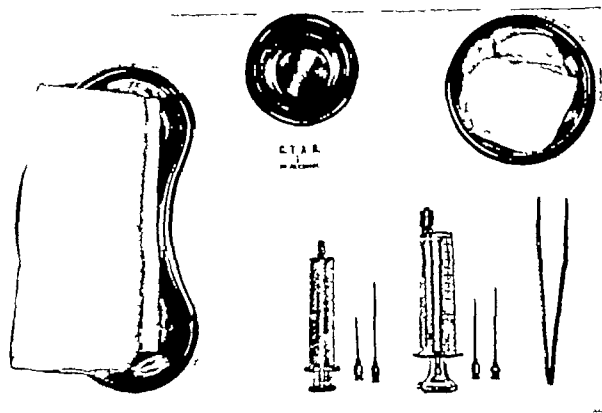


Fig. 4 Tray for intercostal nerve block.

TRAY FOR MANAGEMENT OF FRACTURED RIBS (FIG 4)

*Sterile Equipment*

- antiseptic solution, e.g. cetavalon in spirit
- dressing forceps and scissors
- 2 10-ml. syringes
- 2 fine, short needles
- 2 1 1/2 inch needles, 15 and 17 gauge
- 2 3-inch needles
- swabs
- procaine solution 1 per cent, 20 ml.
- long-acting local anesthetic solution, 6 to 10 ml.
- ampules

*Unsterile Equipment*

- collodion
- absorbent cotton
- elastoplast 3 inches wide
- scissors

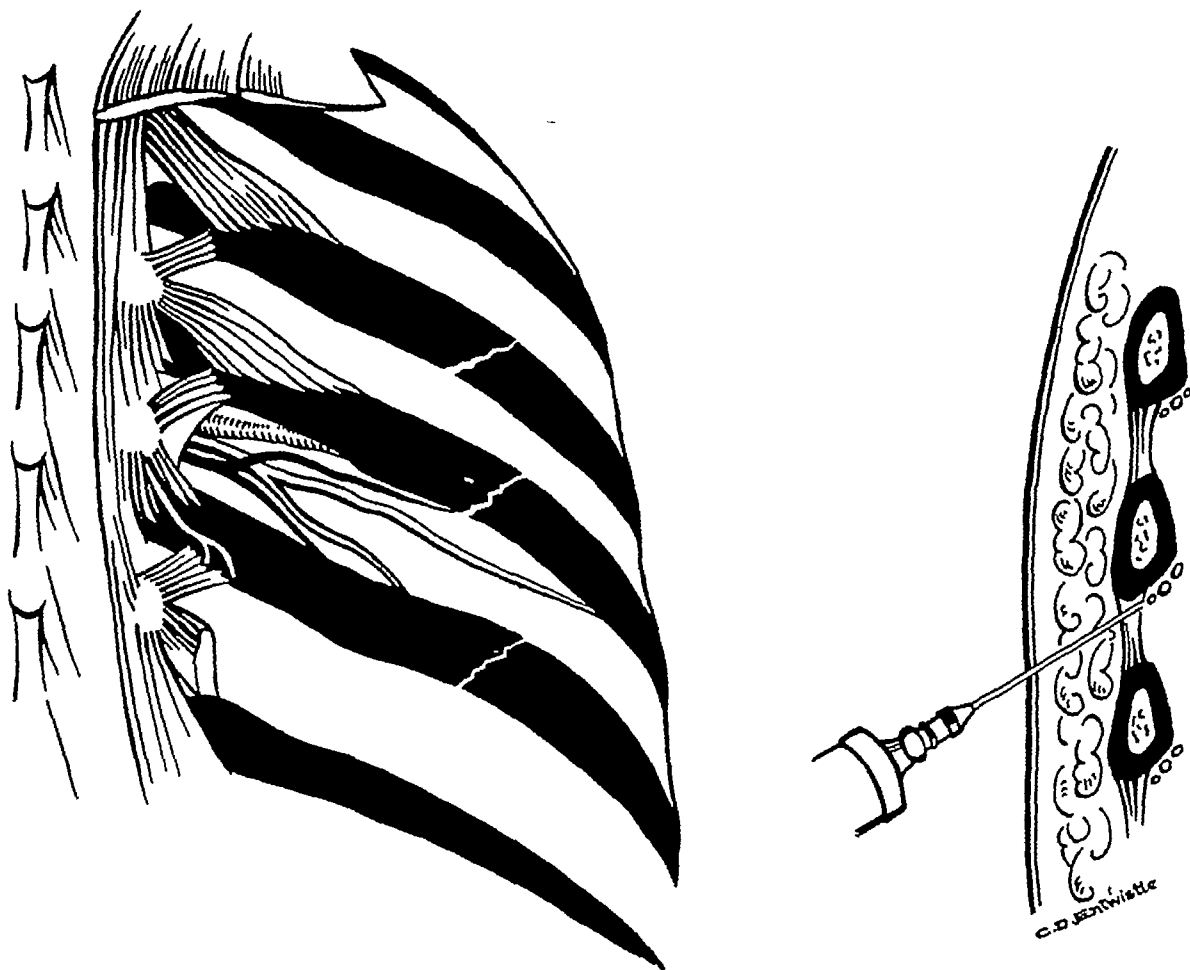


Fig 5A. Anatomy of the paravertebral region and intercostal space

Fig 5B Technic of intercostal nerve block

The needle is advanced to the lower border of the ribs, withdrawn a fraction of an inch, and then advanced into the subcostal groove where the local anaesthetic is injected. Care should be taken not to enter the pleural cavity especially if the syringe is detached.

**Technic. Position.** It is more convenient if the patient can be sat up and supported on a cardiac table, especially if the fractures are bilateral. If the patient is too ill, he is turned on his side with knees drawn up as for lumbar puncture.

**Infiltration.** Procaine skin wheals are raised just lateral to the vertebral transverse processes and directly behind the lower border of the fractured ribs. The needle is advanced to the ribs, withdrawn a fraction of an inch, and then advanced again to a point immediately beneath the rib in the subcostal groove, where 5 ml of 1 per cent procaine are injected (Fig. 5B). As there is an intercommunication between intercostal nerves, the normal rib spaces above and below the fracture are similarly injected. It is not necessary to infiltrate the fracture site, though it is of value where the patient cannot easily be prepared for posterior intercostal nerve block.

As the local anesthetic takes effect, pleural pain disappears and the patient can breathe and cough in comfort. To prolong this effect, a long-acting local anesthetic solution (such as procaine base 1.5 per cent, butyl p aminobenzoate 6 per cent, benzyl alcohol 5 per cent in sterile almond oil) is next injected through the same skin punctures. As the mixture is very viscous, a wide-bore 15 or 17 gauge needle is recommended. One such treatment usually suffices.

If the patient still has difficulty in raising his sputum or if x-ray film confirms the presence of a collapsed lobe or lung, aspiration bronchoscopy is then performed. This is most conveniently done with a portable bronchoscope and suction, and with the patient sitting up in bed (see Chapter 2).

Thus, by intercostal nerve block and bronchoscopic aspiration, the vicious circle of fear of coughing from pain, sputum retention, atelectasis and aggravation of respiratory distress leading to pneumonia is effectively broken.

### MANAGEMENT OF STERNAL FRACTURES

When there is no displacement and no evidence of chest wall and intrathoracic complications, bed rest for two weeks alone is required, by the end of which time the fracture will be firm.

Scudder (14) describes how *overriding* fragments may be reduced by placing the patient on his back, extending his head over the end of a table, next raising his arms above his head, and rotating them slowly and firmly outward, during which time the assistant applies counter traction to his body. An attempt should be made to maintain reduction by placing a firm pad over the line of fracture and fixing it in position with elastoplast.

The staved-in sternum further described in Chapter 5 can be readily elevated by screwing in a cup hook or passing a stainless steel wire around it with a Gilliam needle and thereafter supporting the sternum with a two-pound weight passed over pulleys on a Balkan beam. Operative treatment may be required—an incision is made and the fragments are reduced and held in place with stainless steel sutures.

If however there is a *compound fracture* with accompanying hemopneumothorax, then operative treatment as described below under "Complications" is required.

### COMPLICATIONS AND THEIR MANAGEMENT

The complications of fractured ribs and sternum are important, since they account for most of the symptoms, signs and dangers. The complications are grouped pathologically as follows: chest wall, pleural, pulmonary, cardiac, diaphragmatic, esophageal, aortic, and abdominal lesions. The most common lesions are *hemothorax*, *surgical emphysema* and *pneumothorax*. Knoepf (15) reviewing earlier work, re-emphasized that mortality occurred only in the group with complications, especially those with lung involvement.

In a review of 730 consecutive cases Rapport and associates (16) found complications in 28 per cent of the total, or in 41 per cent of the 490 patients admitted to the hospital. The complications were grouped as follows:

1	Chest wall	17.9%
2	Pleural	26.5%
3	Pulmonary	32.6%
4	Mediastinal	1.6%
5	Abdominal	11.8%

### CHEST WALL COMPLICATIONS

There are three chest wall complications, namely *surgical emphysema*, *flail chest*, and the rarer *compound fractures*.

*Surgical emphysema* may at first be localized. At times, however, it may be widespread, extending to the eyelids, hands, scrotum and feet (Fig. 6). In essence a torn parietal pleura allows the escape of air from the pleural cavity and of adherent



Fig 6 Gross surgical emphysema

lung into the subcutaneous tissue. Emphysema can, however, act as a safety valve, even averting death from tension pneumothorax.

*Mediastinal emphysema* is a serious complication both because mediastinal tension prevents adequate filling of the great veins and the heart, and because of the danger of infection. Clinically, it can be detected by auscultation, revealing a pericardial crunching sound synchronous with the heart beat. Later, suprasternal crepitus appears and, as it progresses, the signs of pressure on the great veins appear and lead to circulatory failure, shock and collapse (see Chapter 20).

*Flail chest* with multiple fractures produces paradoxical respiration, thus seriously interfering with the bellows action of the chest wall (see Chapter 5).

*Compound fractures* that communicate with the pleural cavity are the more serious, on account of the accompanying hemopneumothorax.

**Management of Chest-wall Complications. Surgical Emphysema** If *localized and static*, surgical emphysema may be ignored. If *localized but extending*, a pad and firm elastoplast support will prevent any further extension. If *generalized and uncomfortable*, it can be relieved by painting the skin with antiseptic lotion, and puncturing with a sharp pointed scalpel, e.g. Bard Parker, No. 11 blade. If *severe and mediastinal* and associated with a pneumothorax that is showing no response to an intrapleural Malecot catheter water-seal drainage, the possibility of a major lung laceration or even tracheal or bronchial rupture must be seriously considered, and thoracotomy is advised.

The management of flail chest is described in Chapter 5. The management of compound fractures is detailed under pneumothorax below.

## PLEURAL COMPLICATIONS

There are three pleural complications namely pneumothorax hemothorax, and pleural effusion

**Pneumothorax** This may be open closed, or tension in type It commonly arises when the fractured rib ends penetrate the lung It is also a sequel of compound chest wall fractures the author has seen it in a football player who had been kicked on the chest wall without any detectable rib fractures Crutcher and Nolen (17) reporting three other such cases feel that this supports Lindskog and Liebow's (18) contention that nonpenetrating trauma may produce pneumothorax by bruising injuries to the lung especially when the force is applied to an expanded lung with closed glottis

If the air leak is valvular a *tension pneumothorax* develops with shift of the mediastinum toward the unaffected side steady reduction of functioning lung volume and, when unrelieved death from asphyxia

**Hemothorax** results from rupture of an intercostal or internal mammary artery torn pleural adhesions, lacerated lung, or rarely leaking aorta. The result is collapse of the underlying lung and progressive exsanguination

**Pleural effusion** may be secondary to the pleural irritation of fractured rib ends

**Management of Pleural Complications. Closed Pneumothorax** The immediate question regarding this complication is "Is there a tension pneumothorax as well?" The presence of both types of pneumothorax is suggested by severe respiratory distress and the position of the heart and trachea and it is confirmed by measuring the intrapleural pressure with a pneumothorax apparatus and a needle inserted through the second intercostal space anteriorly It can be immediately relieved by inserting an open intercostal needle or trocar and cannula (Chapter 2) It is however more effectively treated by an intercostal Malecot catheter and water-seal drainage

Inasmuch as a *small uncomplicated pneumothorax* will slowly absorb in the absence of respiratory distress it may be observed for 24 to 48 hours and then rechecked with roentgenograms

If the *pneumothorax is large or increasing* it is unlikely to be controlled by aspiration alone In fact, when continued aspiration of air leaves the pneumothorax at atmospheric or positive pressure a persisting lung fistula is *confirmed* Water-seal drainage and continuous suction with an efficient pump are the only sure way to remove the air and to allow the lung to re-expand and reach the parietal pleura. Thereafter the fistula usually closes (see Chapter 2)

If *this measure fails and a large pneumothorax persists* for more than 24 hours then the presence of a bronchial rupture or a gross tear of the lung is strongly suspected. Bronchoscopy may reveal a bronchial rupture but not a lung laceration. Both conditions require thoracotomy and suture Thompson and Eaton (19) reported successful early repair of a rupture of the intrathoracic trachea complicated by avulsion of the right upper-lobe bronchus, using interrupted 00 silk sutures

**Open Pneumothorax and Compound Fractures** In treating compound fractures the practitioner is constantly aware that *open pneumothorax is the principal danger* These compound fractures most commonly occur as a sequel to automobile accidents, gunshot wounds falls from trees, and the like The affected lung is collapsed thus aggravating respiratory distress

The pneumothorax is temporarily controlled and air excluded by applying to the wound a pad soaked in antiseptic solution or a sterile petrolatum jelly pack, covering this with a large cotton pad, and strapping firmly to the chest wall with elastoplast.



As a further first-aid measure, when the wound is small, it has also been possible to plug the opening temporarily by grasping the lung substance with forceps and drawing it into the wound

The patient is immediately admitted to hospital, taken to the operating room, and anesthetized and intubated with a cuffed endotracheal tube. Thereafter, exploratory thoracotomy and débridement and suture of lung, other viscera, and chest wall can be safely performed. The chest wall is closed over water-seal drainage, and strong suction is applied to remove air and to achieve complete re-expansion of the lung.

Twenty-four hours later, the chest is x-rayed. If the lung is fully re-expanded and all fistulas closed, the intercostal tube is removed. If the pneumothorax is still present but the tube is blocked, it is changed on a Malecot catheter introducer and a further check made next day. This may need to be repeated several times until re-expansion of the lung is complete. The patient receives antibiotics—penicillin, one million units daily and streptomycin 1 gm daily as “cristomycin,”—for four or five days after operation. If convalescence is then uncomplicated, antibiotic therapy is stopped.

It is rare for such a wound, properly treated, to lead to empyema; but, if it does, then the principles and methods of treating empyema apply.

**CASE REPORT COMPOUND FRACTURE OF THE STERNUM** On December 18, 1955, a boy aged 10 years was struck by his father's truck while riding his bicycle up a driveway. He received a sucking pneumothorax with a large wound across the front of the chest. As a first-aid measure, this was covered by a “shell dressing” firmly strapped to the chest. He was transported 30 miles to a hospital by ambulance, and the physician who attended him at the scene of the accident wisely telephoned ahead to warn the thoracic surgeon of his approach.

On admission, he was taken directly to the operating suite where everything was in readiness for thoracotomy. Anesthesia was induced, using oxygen and cyclopropane. A cuffed endotracheal tube was inserted into the trachea and inflated. Thus, the pneumothorax was controlled and the lad made safe. His clothing was then removed.

When the shell dressing was lifted, a wound seven inches long and three inches wide was found across the front of the chest, on both the right and left of the midline. There was a transverse compound dislocation of the sternum at the level of the sternal angle. On the left side, the second and third costal cartilages were dislocated from the sternum, and there was a sucking pneumothorax of the left pleural cavity. Bleeding from the left internal mammary artery had caused a large left hemothorax. The right pleural cavity had escaped injury.

The wound was painted with antiseptic solution (cetavlon in alcohol), towels applied, and the left pleural cavity inspected. It was found to contain at least 150 ml of blood and clots. These were removed and an intercostal drainage tube inserted in the fourth left intercostal space and attached to a water-seal bottle. The sternal fragments were drilled and united by three stainless steel sutures, the wires being threaded through a Gilliam needle. After the sutures were tied, the sternal fracture appeared firm. The second right costal cartilage and the second and third left costal cartilages were sewed to the sternum with chromic catgut sutures. The pectoral muscle fibers were next reapproximated and the skin closed with interrupted nylon sutures. A firm pad and elastoplast cover were applied to the wound. During the course of the operation, the patient had a one-pint blood transfusion.

An immediate postoperative x-ray film confirmed that the left pneumothorax had almost completely disappeared. The skull was also x-rayed, but no cranial lesions were detected. Two hours later, the boy's condition had greatly improved and he was talking and drinking fluids. He returned to school in February 1956, and he remains well.

**Hemothorax** Beside being the most common complication, hemothorax is the most serious but it usually ceases before blood loss is great. Occasionally, however bleeding may continue and fill the pleural cavity with clots and serum. The underlying lung collapses and becomes functionless. Diagnostic aspiration confirms the presence of blood.

*When hemothorax is detected early* half hourly x ray films are taken with the patient supported in the upright position in order to check the rate of bleeding. If the serial films reveal the fluid level to have risen one or more intercostal spaces, the bleeding is continuing and emergency thoracotomy is required.

*If the hematoma is small* basal, and unaccompanied by respiratory distress early and complete aspiration is the aim. It should however be remembered that when the blood has clotted, only half the x ray "shadow" can be aspirated, namely the serum. The clot must then be liquefied with enzymes such as varidase and aspirated 24 hours later. Varidase itself is not without some disadvantages, as (a) a rind of fibrin may remain on the lung to keep it permanently collapsed unless formally decorticated (b) the fever and accompanying tightness in the chest are an annoyance to many patients, and (c) repeated aspirations may introduce staphylococci and produce an unwelcome empyema. Aspiration is of value only for a small hemothorax.

*If however the hematoma is large* and the pleural cavity more than a third full of clot, or if organization has occurred, it is useless to temporize. Early thoracotomy and evacuation of the blood clot with water-seal drainage allow full re-expansion of the lung with minimal convalescence. At the same time the chest wall can be stabilized with chromic catgut or stainless steel sutures (see Chapter 5).

*The guiding principle of removing blood clot to avoid empyema and to restore lung function remains the key to successful management of hemothorax. Surgical removal gives the best results with least residual disability and quickest recovery (20)*

**Pleural Effusion** Rarely a pleural effusion accompanied by fibrin clot develops some days after the ribs are fractured. In the following case, chest aspiration proved fruitless until the fibrin clot was liquefied by varidase.

**CASE REPORT FRACTURED RIBS CAUSING PLEURAL EFFUSION** On June 9 1955 W. B., a male aged 72 years was admitted to a county hospital 24 hours after a heavy fall from his horse onto his left side and shoulder. Chest films showed fractures of the left fourth, fifth, sixth, and tenth ribs. At first, he improved with rest, chemotherapy and postural drainage.

On June 20 he became febrile and developed a large left pleural effusion.

On June 30 he was transferred to a base hospital where chest films and chest aspiration confirmed left pleural effusion which consisted of clear straw-colored fluid with a protein content of 3.7 per cent. Only 4 oz. could be aspirated.

July 4 because the effusion persisted, varidase was injected into the left pleural cavity. Twenty four hours later upon a further chest aspiration, 46 oz. of sterile, straw-colored pleural fluid were removed. Chest x ray films thereafter showed a completely expanded left lung, and the patient was discharged on July 15. Study of the films showed that the ribs were not in exact alignment. It was surmised that their sharp ends had damaged the underlying lung and caused the pleural effusion.

## PULMONARY COMPLICATIONS

There are four pulmonary complications: laceration, contusion, atelectasis from sputum retention, and ruptured trachea or bronchus.

**Lacerations of the Lung** Lung lacerations caused by fractured rib ends may be

small or large, single or multiple, superficial or deep. There is invariably an accompanying hemopneumothorax.

*Contusion of the Lung with Lung Collapse.* Pathologically, in this condition the lung parenchyma and alveoli are disrupted with blood. Blood-stained sputum is the rule and, because of shock, pain, flail chest, and rapidly increasing bleeding, the patient may be unable to clear the sputum. Since the sputum lies in the trachea and bronchi, these passages become blocked, the air behind the blood is absorbed, the corresponding lung tissue becomes *atelectatic*, and the heart and mediastinum are forced toward that side by atmospheric pressure overdistending the still-functioning pulmonary segments. The patient is in immediate distress, has a sense of tightness in the chest, and physiologically experiences relative anoxia. If both main bronchi are blocked, there is complete anoxia and death occurs within three minutes. *The essential problem is, therefore, sputum removal to relieve anoxia and restore lung function.*

*Traumatic Rupture of the Trachea or Bronchi.* This type of rupture is accompanied by subcutaneous and mediastinal emphysema as well as pneumothorax. The lung segment beyond the rupture collapses and remains functionless until treated surgically.

**Management of Pulmonary Complications.** *Laceration of the Lung.* Air invariably escapes from the lacerated lung and is discovered as surgical emphysema or pneumothorax.

*If the lacerations are small*, provided the accompanying pneumothorax is treated by intercostal water-seal drainage and suction, these lacerations usually seal themselves within two days. *If the laceration is large* and the pneumothorax fails to respond to the above measures, and if other lesions are suspected at the time of injury, then thoracotomy and lung suture are the only satisfactory treatment. Not only should the lungs be inspected, but also the possibility of bronchial or tracheal rupture should be borne in mind and looked for, especially in the presence of mediastinal emphysema.

*Lung Contusion and Sputum Retention.* One of these is the sequel to the other, especially if effective coughing is further prevented by pain or a flail chest wall. If there is the least sign of retention of sputum, with or without lung collapse and especially persisting after physiotherapy, then emergency bronchoscopy is required forthwith.

If bronchoscopy is repeatedly required or if there is sputum retention in a patient with a fractured jaw that prevents bronchoscopy, then *tracheotomy must be performed immediately*. Thereafter, sputum is easily aspirated by the special nurse, the airway is kept clear, the dead space is lessened, and the patient's convalescence is made smoother.

*Ruptured Trachea and Bronchus.* Treatment of these complications depends on the following:

1. The time-lag between rupture and discovery,
2. The extent of the rupture,
3. The extent of distal lung damage.

These points can be clarified only by bronchoscopy and thoracotomy. *If rupture is discovered within three days of injury* and if the fracture is clean and the lung beyond is not infected, then suture with fine No. 2 silk sutures is required. The chest wall is

closed over two water seal tubes one as an air vent and one as a drain for pleural fluid. Suction must be applied to both tubes to encourage full lung expansion. Antibiotic therapy such as penicillin one million units per day and streptomycin 1 gm per day, is given by injection, and the state of the expanded lobe is watched by daily roentgenograms. The tubes are removed when they cease to function. Final bronchoscopy and bronchography two months later should confirm a satisfactory result.

If rupture is discovered two or more weeks after injury when persisting lobar collapse has necessitated bronchoscopy and a stricture is revealed at the site of the bronchial rupture then thoracotomy is indicated. The definitive treatment—be it excision of the structure and resuture or resection of the functionless lung segment—depends on the findings. Age appears to be no bar for in 1955 Ellis and associates (21) successfully repaired, by end-to-end anastomosis, a completely severed right main bronchus which a three year-old child had had for a week. In Thompson and Eaton's case (19) a completely severed left main bronchus was satisfactorily anastomosed two months after injury with restoration of lung function.

### CARDIAC COMPLICATIONS

These include *traumatic asphyxia hemopericardium* from cardiac wounds and *traumatic myocardial infarction*. In *traumatic asphyxia* owing to a sudden blow on the chest, blood is forced back up the superior vena caval system with multiple rupturing of the subcutaneous capillaries of head, neck, arms and conjunctivas. As Souttar (22) points out, it is remarkable that the slightest local pressure prevents the discoloration so that the lines of suspenders, shirt collar, and hat rim stand out as white bands on a blue background.

*Hemopericardium* may cause death from massive hemorrhage or tamponade. As little as 200 ml. of blood may cause fatal tamponade if it escapes rapidly into the pericardium (23). These complications are further discussed in Chapter 22.

*Traumatic myocardial infarction* is rare and is usually an autopsy finding.

### DIAPHRAGMATIC COMPLICATIONS

See Chapters 5 and 21

### ESOPHAGEAL AND AORTIC RUPTURE

See Chapters 5 and 18

### ABDOMINAL COMPLICATIONS

Ruptured liver, spleen, and kidneys may accompany fracture of the lower ribs. These ruptures are discussed in Chapter 5.

### CONCLUSIONS

To summarize then, the usual treatment of fractured ribs embodies

1. Intercostal nerve block to relieve the pain of respiration
2. Intercostal catheter drainage to remove the pneumothorax and any fluid in the pleural cavity
3. Aspiration bronchoscopy to remove the sputum and allow re-expansion of the lung

- 4 Support of a flail chest;
5. Prompt recognition and correction of chest-wall, pleural, and pulmonary complications.

Pain is the chief cause of sputum retention. Only by application of these five principles of treatment can the vicious connection between pain and sputum retention be broken.

#### REFERENCES

- 1 Watson-Jones, R. *Fractures and Joint Injuries*, 4th ed., Edinburgh, E & S Livingstone, Ltd., 1955.
- 2 Jones, H. E. Rib fractured by coughing, *Glasgow M J*, 61:118, 1904.
- 3 Kleiner, S. B. Fracture of ribs by muscle action, with report of a case, *Boston M & S J*, 190:1034, 1924.
- 4 Palfrey, F. W. Fracture of ribs by muscular action, *Boston M & S J*, 191:498, 1924.
- 5 Swineford, D., and McKinnon, J. Multiple fractures of ribs by cough. Report of a case, *Ann Int Med*, 23:442, 1945.
- 6 Paulley, J. W., Lees, D. H., and Pearson, A. C. Cough fractures in late pregnancy, *Brit M J*, 1:135, 1949.
- 7 Hinton, D., and Steiner, C. A. Fracture of the ribs, *J Bone & Joint Surg*, 22:597, 1940.
- 8 Holderman, H. H. Fracture and dislocation of the sternum. Report of three cases, *Ann Surg*, 88:252, 1928.
9. Gurlt, E. *Handbuch der Lehre von den Knochen Bruch*, Hamm, G. Grote, 1864, vol. 2, Pt. 1, 264. Quoted by Holderman (ref. 8).
- 10 Stuck, W. G. Fracture of the sternum and thyroid cartilage, *Am J Surg*, 38:561, 1937.
- 11 Latteri, S. L'alcoolizzazione dei nervi intercostali nella cura delle fratture delle costole, *Riv San Siciliana*, 21:249, 1933. Quoted by Burford, T. H., and Burbank, B., Traumatic lung. Observations on certain physiologic fundamentals of thoracic trauma, *J Thoracic Surg*, 14:415, 1945.
- 12 Rovenstine, E. A., and Byrd, M. L. Use of regional nerve block during treatment for fractured ribs, *Am J Surg*, 46:303, 1939.
- 13 Harmon, P. H., Baker, D. R., and Kornegay, R. D. Uncomplicated fractures of ribs and major injuries to the chest wall, *J A.M.A.*, 118:30, 1942.
- 14 Scudder, C. L. *The Treatment of Fractures*, 9th ed., Philadelphia, W. B. Saunders Co., 129, 1923.
- 15 Knoepp, L. F. Fractures of the Ribs—A review of 386 cases, *Am J Surg*, 52:405, 1941.
- 16 Rapport, R. L., Allen, R. B., and Curry, G. J. The fractured rib—a significant injury. An analysis of 730 consecutive cases, *Arch Surg*, 71:7, 1955.
17. Crutcher, R. R., and Nolen, T. M. Traumatic pneumothorax without rib fracture, *J Thoracic Surg*, 29:621, 1955.
- 18 Lindskog, G. E., and Liebow, A. A. *Thoracic Surgery and Related Pathology*, New York, Appleton-Century-Crofts, Inc., 1953.
- 19 Thompson, J. V., and Eaton, E. R. Intrathoracic rupture of the trachea and major bronchi due to crushing injury, *J Thoracic Surg*, 29:260, 1955.
- 20 Borrie, J. Emergency thoracotomy for massive spontaneous hemopneumothorax, *Brit M J*, 2:16, 1953.
- 21 Ellis, F. H., Andersen, H. A., and Hayles, A. B. Complete traumatic rupture of the bronchus with successful surgical repair. Report of a case in a 3-year-old child, *Proc Staff Meet, Mayo Clin*, 30:268, 1955.
- 22 Souttar, H. S. *The Art of Surgery*, 4th ed., London, W. Heinemann, Ltd., 1940, vol. 2.
- 23 Griswold, R. A., and Maguire, C. H. Penetrating wounds of the heart and pericardium, *Surg, Gynec & Obst*, 74:406, 1942.

## CRUSH INJURIES AND CHEST WOUNDS

The principles of managing major thoracic trauma differ in no way from those governing fractures of one to three ribs but, as the effects of major injury are more severe and extensive the management is more exacting. This chapter is concerned with patients who have *more than three fractured ribs*.

### PATHOLOGY

Most patients in this category are men in the 20 to 40 age group. The frequency of serious injury is emphasized in a series of 30 chest injuries referred to the Dunedin Thoracic Surgical Department in the past 3 years. Ten had met with violent accidents of these 6 died 3 within 6 hours 2 within 19 hours and the last after 3 days.

The wide variety of causes included a fall from a motorcycle crashing off the road in a van being crushed between two vehicles being struck down by a passing train or car and being tossed by a bull. To this list are added gunshot wounds or knife injuries of war or peace. As Bernatz and Kirklin (1) stress the high mortality rate attending these severe injuries is attributable to serious derangement of cardio-respiratory function.

The exact pathologic lesion not always obvious, varies greatly in extent and severity and is usually proportional to the severity of the trauma. The ribs lung heart, and aortic injuries have already been described.

Major trauma, in addition, produces a traumatic wet lung in which there is an increased quantity of interstitial and intraalveolar fluid with increased fluid in the bronchopulmonary tree and increased difficulty in coughing it up (2).

The diaphragmatic lesion is a traumatic rupture of the dome producing herniation of abdominal contents, usually into the left pleural cavity. It may at first be overlooked, misinterpreted on subsequent roentgenograms as "raised diaphragm" and only later found to be present when acute or chronic gastric symptoms demand complete investigation (3) (see Chapter 21).

Abdominal lesions which occur are rupture of the solid viscera, liver kidneys spleen, and rarely perforation of the bowel.

The wide variety of serious lesions which clinically appear trivial and even allow several hours of life is shown in the following case history.

**CASE REPORT MULTIPLE THORACIC INJURIES** J. H. a male aged 25 years, on November 25 1952, lost control of a fully loaded gasoline truck, charged down a steep street at 80 miles per hour and crashed through a factory wall. He was briefly pinned between the steering wheel and the seat. He was promptly admitted to hospital and, despite a flail chest, appeared to be in surprisingly good condition. There were bilateral fractures of the fourth to eighth ribs, with a small hemothorax (Fig. 1). Immediate treatment included blood transfusion, bilateral water-seal drainage, intercostal injections of a long-acting local anesthetic, and support of his flail chest with cotton pads and adhesive plaster with apparent relief. He died suddenly three hours after admission, when thoracotomy was being considered.

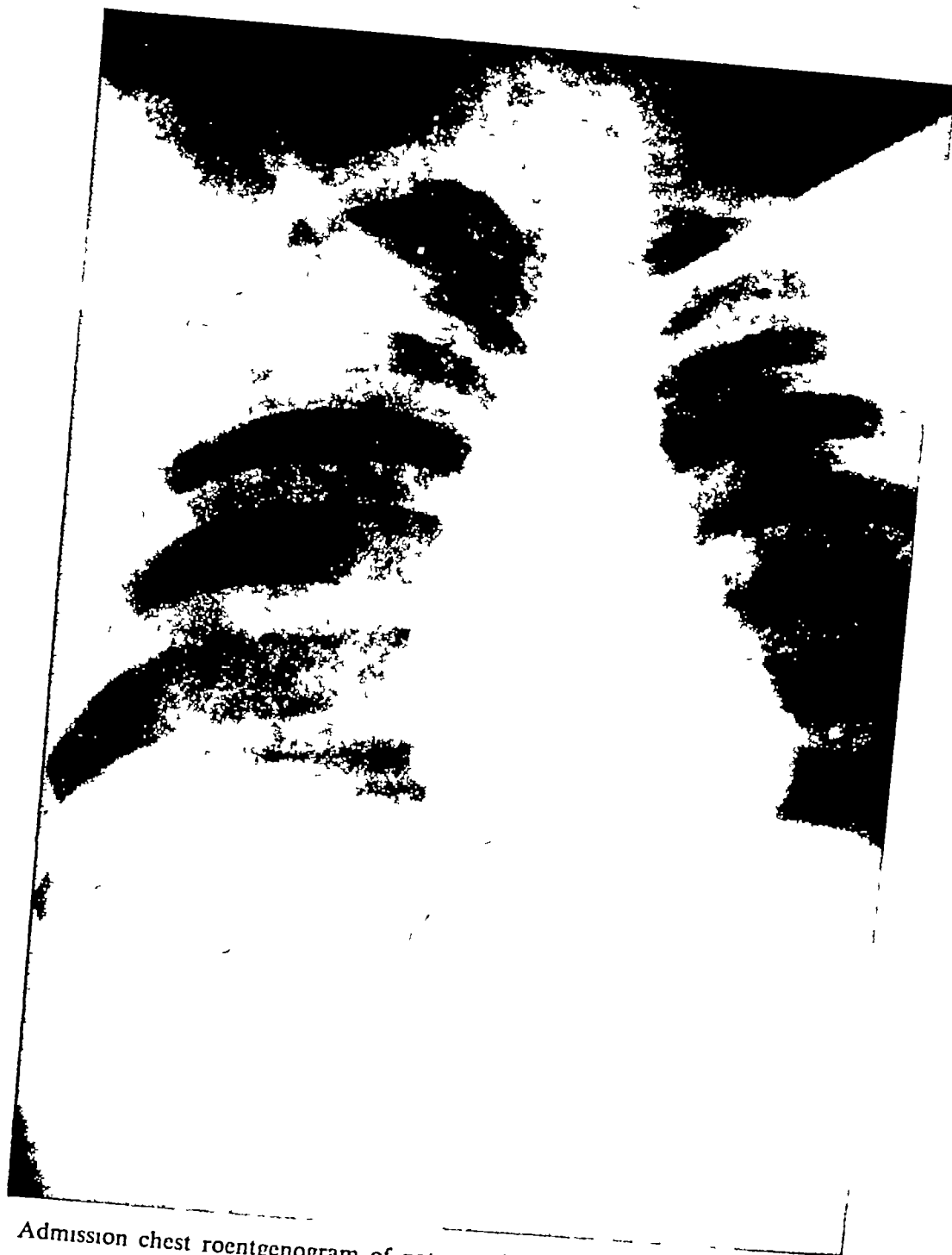


Fig 1 Admission chest roentgenogram of patient who crashed gasoline truck through brick wall at 80 miles per hour

## Clinical Features

*Autopsy* showed trachea and bronchi obstructed by blood and confirmed the fractured ribs. There was a bilateral hemothorax, ruptures of both domes of the diaphragm, the esophagus, the aorta, the liver, the spleen, and the left kidney as well as a mediastinal hematoma and fractured pelvis.

With the many associated injuries e.g. of abdomen, spine, spinal cord, head, and limbs, a patient is frequently and understandably admitted to the care of a general or orthopedic surgeon. While the patient must be treated as a whole, *the rapidity with which asphyxia kills makes it imperative to give priority to the thoracic lesions* of which injury to the lung is the most insidious and most lethal. For the result of severe chest trauma is loss of ventilatory lung function from flail chest, blocked bronchi, and disordered pleural cavity and also loss of parenchymal lung function from contusion of the lung.

### CLINICAL FEATURES

Patients with crush injuries and chest wounds fall into four clinical groups:

- 1 Those so severely injured that they never reach the hospital
- 2 Those with relatively mild injuries
- 3 Those with severe injury and closed pneumothorax
- 4 Those with open pneumothorax

In all these groups complacency is unwarranted. Each case demands respect, immediate critical assessment, and constant vigilance.

Patients with open pneumothorax are often more satisfactory to treat in that the very urgency of their condition demands immediate operative relief. To decide whether the patient with a closed pneumothorax is mildly or severely injured is *the real problem*. Even the gravely ill patient may at first sight appear to have recoverable lesions. At times this distinction proves difficult and disappointing. The following points are of assistance in assessing these cases:

**History.** The practitioner can attempt to gauge the severity of the blow and the possible pathologic state if a history can be obtained.

**Physical Examination.** This usually shows surgical emphysema, flail chest, and pneumothorax, and sometimes tracheal deviation and respiratory difficulty. *Heavily blood stained sputum is of serious significance.*

Where there has been a penetrating wound the course of the projectile is best determined by considering the external wound, identifying the fractured ribs, visualizing the metallic fragment (when present), and reconstructing the patient's position at the time of wounding (4-5).

The abdomen must be carefully examined in all thoracic casualties. Tenderness and rigidity over the liver, spleen, or loins combined with signs of blood loss—namely increasing pallor and sweating, rising pulse rate, and falling blood pressure—indicate associated injury to these organs. Repeated specimens of urine should be examined for blood staining. *Acute dilatation of the stomach* may also occur with a purely thoracic wound, accompanied by abdominal pain, tenderness, and rigidity and a silent abdomen.

Regarding differentiation, when the lesion is thoracic the pain is unilateral, less obvious on inspiration, and largely relieved by intercostal nerve block. However when the lesion is abdominal some degree of spasm, rigidity, and tenderness upon deep pressure usually remain.



**Roentgenograms.** A straight chest film taken with the patient lying flat is often disappointing, the hemothorax masking the degree of pneumothorax and often the full extent of the *rib fractures* as well. When, however, the patient can be supported semireclining or sitting up, the proportion of hemothorax to pneumothorax is far more clearly defined. *Sternal fractures* can be adequately shown only by lateral films.

A mouthful of radiopaque fluid swallowed before the chest films are taken may reveal the presence of an otherwise *unsuspected esophageal perforation*. By this means, or by swallowing a Levin tube, the practitioner can demonstrate that a suspected raised diaphragm is in fact a *ruptured diaphragm*. An increasing or unusual mediastinal shadow may also be a *traumatic aortic aneurysm* or *mediastinal hematoma*.

If the question of continued intrathoracic bleeding is raised, roentgenograms must be repeated at half-hourly or hourly intervals. If there is the least doubt about an abdominal lesion, an abdominal film also is required.

In these patients, however, the taking of roentgenograms must not be made an end in itself, nor should roentgenograms take priority over a real emergency such as clearing a blocked airway (6). X-rays are of little value in assessing the seriousness of the injury and the imminence of death and have been known to mislead even the experienced.

**Further Assessment.** The history, clinical findings, and roentgenograms usually suffice for diagnosis, but the exact state of the patient can often be determined only by *continued observation*. When carried out with the help of a special nurse who records pulse, respiration rate, and blood pressure at five-minute intervals, and with judicious repeat skiagrams, continued observation will materially assist in recognition of advancing lesions, especially when intrapleural bleeding, tension pneumothorax, or a bronchial obstruction are insidiously progressing.

### GENERAL PRINCIPLES OF MANAGEMENT

The fundamental problem in all major thoracic trauma is to correct the disordered chest function. Though some patients may be doomed, by strictly observing the following principles of management many can be saved.

The *six essentials* are to:

1. Secure the airway and administer oxygen;
2. Relieve intrapleural tension;
3. Relieve pain,
4. Stabilize the chest wall,
5. Give blood as required,
6. Explore the chest when indicated.

**The Airway and Oxygen.** The airway commences at the nose and ends at the pulmonary alveoli. For oxygen administration to be effective, there must be (a) an efficient method of supply (such as nasal catheters or a plastic mask); and (b) a clear airway.

**Bronchoscopy** When blocked, an airway can be adequately secured only by endobronchial suction, via either a bronchoscope or the tracheotomy tube. In serious cases, lesser procedures such as blind, intermittent transnasal or oral aspirations are worthless. Repeated bronchoscopic aspiration is also unsatisfactory, as it requires repeated manipulation of the patient, with trauma to the larynx resulting in edema.

Further an emergency bronchoscopy may be required before the bronchoscopist can be summoned.

**Tracheotomy** (See Chapter 2 ) It is permissible in these cases to perform *bronchoscopy* once. But, if there is the least suggestion of re-accumulation of endobronchial secretion, or if the original secretions are thickly blood stained, it is a far safer and sounder practice to do an *immediate tracheotomy*. Tracheotomy is further indicated when there are oral injuries or fractures of the jaw or of the spine.

Tracheotomy is readily performed in the ward with minimal disturbance to the patient and allows of frequent endotracheal aspiration by anyone, above all by the special nurse *who must be in attendance*. In severe cases especially with marked flail chest, an Engstrom type of respirator can be lifesaving, particularly when used with a cuffed tracheotomy tube (7). The Engstrom respirator is a positive pressure resuscitation apparatus.

Carter and Giuseffi (8) point out that beside the mechanical advantages of being able to clear the airway readily through a tracheotomy tube there are two equally important physiologic benefits

- 1 Decreasing the respiratory dead space and thereby increasing effective ventilation
- 2 Decreasing the breathing resistance to inspiration and expiration. Any tendency to progressive mediastinal emphysema is also reduced.

**Relief of Intrapleural Tension.** Tension pneumothorax and hemothorax are promptly relieved by water seal drainage. A Malecot catheter is inserted with a trocar and cannula low down in the midaxillary line. A catheter in the second intercostal space in front and connected to an electric suction device may enhance the effect of the first catheter (see Chapter 2 Figs 3 through 6). However when intrapleural bleeding continues or the pneumothorax is unrelieved, urgent thoracotomy is required.

**Relief of Pain.** With the airway secured and the pleural tension controlled, chest-wall pain is next relieved by paravertebral injection of a long-acting local anesthetic as described in Chapter 4.

*Sedatives should never be given as a sole treatment.* They do relieve pain, but they also depress the cough reflex and encourage in patient and surgeon alike an attitude of unreal complacency until the final moment when the patient, unable to clear his lungs, drowns in his own sputum.

That relief of pain is not of the highest priority in these serious emergencies is well shown by the following case.

**CASE REPORT** W. K., a male aged 30 years, fell from a motorcycle on August 29 1955. Four hours later after an ambulance journey of 100 miles, he was admitted under the care of the emergency surgeon. He was semiconscious, not communicable and pink in color; his breathing was shallow and bubbling and there was a flail chest. A roentgenogram showed fractures of the right clavicle and of the right second to fourth ribs and left second, fifth, and sixth ribs. This film was taken with the patient recumbent, the blood in the pleural cavity was marked. His observers during the night believed him to be in fair condition, though by morning he had surgical emphysema and a respiratory rate of 45 per minute.

At this stage, when semicomatose and cyanosed, he was ...

surgeon He was taken at once to the endoscopy room for aspiration bronchoscopy, but before it was done, an attempt was made to relieve his pain by paravertebral injection of local anesthesia While this was under way, his condition suddenly deteriorated, he was bronchoscoped and the bronchial tree, full of blood and clots, was sucked clear A right pneumothorax was simultaneously relieved by an intercostal water-seal drain He did not respond, and further resuscitative measures, including cardiac compression, were of no avail Autopsy confirmed the immediate cause of death to be asphyxia from contusion of the lungs with bilateral hemothorax and bronchial occlusion

*Comment* The delay in referring this patient proved to be his undoing His flail chest, moist respiration, deepening cyanosis, and inability to cough up sputum were clear indications for emergency action Had he been treated promptly by bronchoscopy, enhanced by tracheotomy, to secure an adequate airway, intercostal drainage, and chest support with pad and elastoplast, the immediate dangerous corner could have been turned to allow of subsequent thoracotomy for hemothorax and chest stabilizing procedures. The final error was to assume that relief of pain took precedence over adequate oxygenation of the lungs

**Stabilizing the Chest Wall.** The *four objectives* for stabilizing the chest wall are

- 1 To restore the elastic continuity of the chest wall,
- 2 To restore negative intrapleural pressure,
- 3 To correct paradoxical respiration,
- 4 To correct mediastinal motion (9)

With the intact chest wall so fundamental a part of the mechanism of normal respiration, a flail chest wall is one of the serious emergencies of traumatic surgery

When there are unilateral rows of fractured ribs (which usually occur in the anterior or posterior axillary lines), the unstable segment lies laterally When there are bilateral fractures (which usually lie in the anterior axillary line or costochondral junction), the unstable segment lies in front Because of muscle and scapular support, posterior rib fractures seldom produce paradoxical respiration

There are three ways of stabilizing a flail chest wall.

1. Pad and strapping support,
- 2 Skeletal traction,
- 3 Surgical fixation

**PAD AND STRAPPING SUPPORT** While intercostal nerve block plus pad and strapping support can relieve a patient with fracture of one or two ribs and are fundamental first-aid measures in controlling a sucking chest wound, they have little place in final treatment of an obviously flail chest wall Regarding strapping support of these serious cases, Bernatz and Kirklin (1) rightly point out that immobilization cannot be satisfactorily accomplished unless both sides of the chest are strapped This in itself limits respiratory excursion, and the effectiveness of the cough is reduced, especially in the aged with poor respiratory reserve At times, too, the skin becomes excoriated at the site, hindering subsequent intercostal nerve block or thoracotomy Nevertheless, this method *has saved lives* when the flail chest was not too severe It is interesting further to note how nature can readjust a severe deformity of the ribs when treated by strapping support, provided the underlying lung is functioning normally (see Chapter 4, Fig 1).





Fig 3A Fracture of sternum



Fig 3B Reduction by cuphook traction

- 5 Apply dressings to the wound,
- 6 Apply up to five pounds traction over a pulley, supported on a Balkan beam.

In an alternate method, Jones and Richardson (11) advise the use of tenaculum forceps to obtain sternal traction. Again, the author has passed wires around the sternum with a Gilliam needle (Fig 4B)

Traction is usually required for 10 to 14 days, by which time the chest wall will be firm once more

**SURGICAL FIXATION** In 1950, Coleman and Coleman (12) first reported a series of 11 patients with multiple rib fractures associated with varying degrees of shock and paradoxical respiration who were treated by internal fixation of the fracture sites by wire. According to their report, disturbed thoracic function was immediately corrected, known and unknown complications were recognized and treated at the time of the operation, and the pain of the fractured ribs was immediately abolished.

*Approach for Anterior Fractures* Fractures of the upper five ribs anterior to the midaxillary line are best approached by an anterior incision along the axillary border of the pectoralis major muscle. Elevation of this muscle will then give excellent access for wiring either the ribs or the costal cartilages (Fig 5)

*Approach for Posterior Fractures* Fractures involving the upper five ribs posterior to the midaxillary line are approached through a pericapsular incision. Frac-



Fig. 4A. Alternative method of applying continuous traction for anterior flail chest wall.

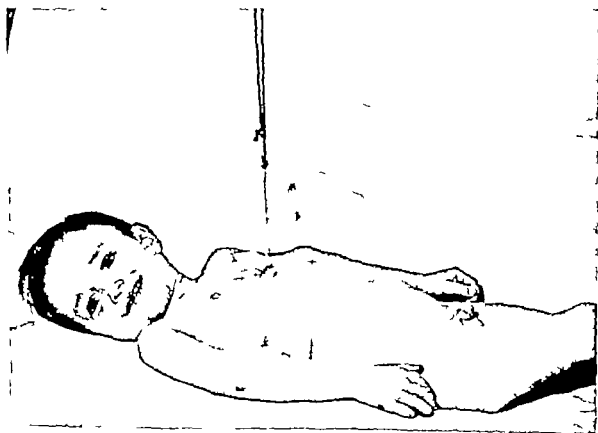


Fig. 4B. Closeup showing detail.

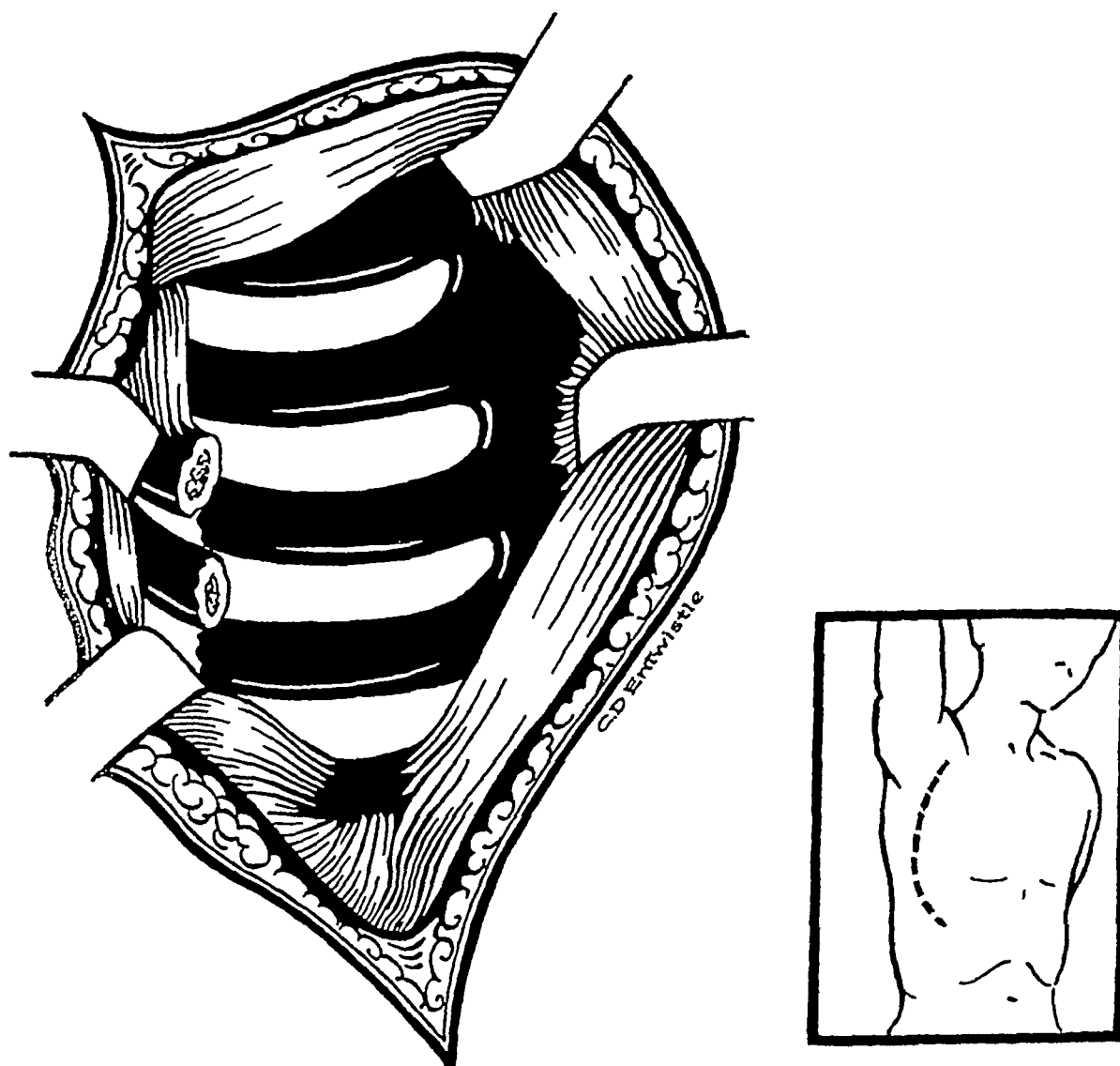


Fig 5 Approach for direct fixation of anterolateral flail chest

tures of the sixth to tenth ribs are exposed by placing the skin incision over the fractured ribs

*Approach for Double Fractures* Excellent exposure is obtained with a U-shaped incision, after which skin, subcutaneous tissue, and latissimus dorsi are reflected upward

A combination of these approaches can be used for exposing fractures at all sites (Fig 6)

*Method of Fixation* To avoid dislocation of the fragments by coughing, ribs must be wired in two planes. The following methods have been found dependable and adaptable to almost any type of rib fracture.

- 1 Oblique fractures require end-on wiring as well as circumferential wiring.
- 2 Transverse fractures require either overlapping of the fragments or end-on fixation over a bone peg

Overlapping of fragments leads to shortening of the rib and is thus not adaptable to multiple fractures in one rib. Intramedullary pegging of transverse rib fractures accompanied by end-on wiring with No. 28 stainless steel wire gives sturdy fixation capable of withstanding respiratory excursion and coughing.

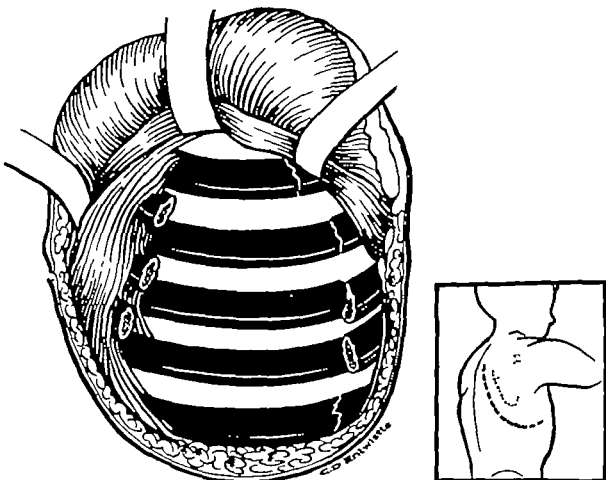


Fig. 6 Method of approaching posterolateral flail chest.



Fig. 7 Three types of rib fixation, left and center with sutures, and, right, with intramedullary bone peg.

The ribs are drilled 2 cm. from the ends of the fragments. A suitable peg of cortical bone 3 cm. long is cut from the superior border of a neighboring healthy rib. After tunneling the medullary cavity of the fracture fragments with a hemostat, the bone peg is introduced and the fragments approximated over the bone graft by drawing the previously placed wire taut. The wire maintains the rib alignment in the longitudinal areas while the bone peg prevents dislocation in the transverse plane. Intercostal muscles sutured around the fractures give additional support.

**Blood Replacement.** After severe chest injuries, shock is the rule and accompanies extravasation of blood into the lung parenchyma or pleural cavity. As bleeding can continue insidiously and as the pleural cavity can readily hold four pints of blood and clot, blood grouping and crossmatching are essential. Any blood loss must be replaced by transfusion.



*On Admission to Hospital* The procedure on admission of the patient to the hospital is as follows, depending on the case:

1. If respiration is seriously embarrassed, emergency tracheotomy and intercostal drainage may be required in the emergency ward. Chest roentgenograms follow and further treatment is carried out. *The time to do a tracheotomy is when considering whether or not it should be done*
2. If there is a compound fracture, the patient should be admitted directly to an anesthetic or resuscitation room adjacent to the operating room. He is then anesthetized and intubated, and the situation is thus brought under control. He is next undressed and prepared for operation
3. When there is a closed injury and the patient is not in acute respiratory distress, chest films are taken and are used as a guide—but *by no means the only guide*—to his lesion. If at all possible, the patient should be sat up, if only to 45 degrees, to allow the shadows of hemo- and pneumothorax to separate. As has been seen, roentgenograms do not answer the most important problem of all. "How serious is the lesion?"
4. Thereafter, if the patient has a bubbling respiration, bronchoscopy, with or without tracheotomy as indicated, is performed. Tension pneumothorax is relieved by intercostal water-seal drainage. Pain is relieved by intercostal nerve block, and the chest wall is supported and transfusion set up.
5. A special nurse and physiotherapist are essential, and the least labored breathing, quickening of the pulse or falling of blood pressure must be reported to the surgeon

As time is paramount in these problems, sterile tracheotomy, intercostal drainage, chest aspirating, and bronchoscopy sets must be kept by the patient's bedside. Such sets should also be standard equipment in all casualty departments.

**Clinical Course.** Any increase in the hemothorax is gauged by increasing pallor, pulse and respiration rates, and by serial chest films at hourly intervals. If bleeding is uncontrollable, if a large hemothorax cannot be aspirated, if a major bronchus or the diaphragm is ruptured, or if the lung is severely lacerated or other lesions are suspected, urgent thoracotomy is indicated.

Since thoracotomy often results in rapid improvement when performed for patients who have compound fractures, and also provides the opportunity for suturing the torn lung and evacuating a hemothorax, it may well be that, with the site of the lesion localized, thoracotomy should be performed for *all* serious closed-chest injuries. Growing experience backed by autopsy findings supports this view.

In country districts where animal or tractor accidents occur, the injured patient may first be admitted to a small country hospital. If, by the above measures, his condition can be improved and made relatively safe, he should be transported as soon as possible to a thoracic unit where all the complexities of persisting pneumothorax, retained secretion, ineffective chest stabilization, and blocked tubes and bottles are more familiar and more readily adjusted. In such a transfer, a doctor should accompany him in the ambulance.

## CONCLUSIONS

Any thoracic trauma is serious. It must be treated with the utmost respect. Many patients recover, but those admitted after major thoracic trauma still remain a major

problem the more so in that their injuries so effectively destroy the mechanism of respiration. The danger is anoxia, and *three minutes of complete anoxia is fatal*

That these patients with major trauma can live several hours after admission to hospital is a *challenge* above all because the victims are often young men and because death is more often based directly on the effects of asphyxia from disorder of chest function than from associated irrecoverable lesions *Time and timing are the essence of the contract*

Shefts' recent monograph (21) gives an excellent account of this important subject.

## REFERENCES

- 1 Bernatz, P. E., Kirklin, J. W., and Olsen, A. M. Severe crush injuries of the chest. Some problems in management, *Proc. Staff Meet., Mayo Clin.* 28 193 1953
- 2 Burford, T. H. and Burbank, B. Traumatic wet lung. Observations on certain physiologic fundamentals of thoracic trauma, *J Thoracic Surg.* 14 415 1945
- 3 Edwards, A. Tudor. Treatment of injuries of the chest, *Brit. M. J.* 2 1096 1938
- 4 O'Rourke, P. V., and Jacobson, L. F. Acute injuries of the diaphragm, *Am. J. Surg.*, 89 769 1955
- 5 Samson, P. C., Burbank, B., Brewer, L. A., and Burford, T. H. Immediate care of the wounded thorax, *J.A.M.A.* 129 606 1945
- 6 Borrie, J. Management of major thoracic trauma, *Australian & New Zealand J. Surg.* 26 229 1957
- 7 Björk, V. O., and Engstrom, C. G., The treatment of ventilatory insufficiency after pulmonary resection and tracheotomy and prolonged artificial ventilation, *J Thoracic Surg.* 30 1955 376
- 8 Carter, B. N., and Glusieff, J. Tracheotomy: a useful procedure in thoracic surgery with particular reference to its employment in crushing injuries of the thorax, *J Thoracic Surg.* 21 495 1951
- 9 Cohen, E. A. Treatment of the flail chest by towel clip traction, *Am. J. Surg.*, 90 517 1955
- 10 Jaslow, I. A. Skeletal traction in treatment of multiple fractures of the thoracic cage, *Am. J. Surg.*, 72 753 1946
- 11 Jones, T. B., and Richardson, E. P. Traction on the sternum in the treatment of multiple fractured ribs, *Surg., Gynec. & Obst.*, 42 283 1926
- 12 Coleman, F. P., and Coleman, C. L. Fracture of ribs—a logical treatment, *Surg. Gynec. & Obst.* 90 129 1950
- 13 Rapport, R. L., Allen, R. B., and Curry, G. J. The fractured rib—a significant injury. An analysis of 730 consecutive cases, *Arch. Surg.*, 71 7 1955
- 14 Cameron, D. A., O'Rourke, P. V., and Burt, C. W. An analysis of the management and complications of multiple (three or more) rib fractures, *Am. J. Surg.*, 78 668 1949
- 15 Ada, A. E. W., and Hevenor, E. P. Reconstruction of defects of the thoracic wall with tantalum mesh gauze, *J Thoracic Surg.*, 21 125 1951
- 16 Rider, W. D. The use of tantalum gauze in repair of large gunshot wound of chest. Report of a case *Brit. M. J.*, 2 1561 1951
- 17 Effler, D. B. Prevention of chest wall defects. Use of tantalum and steel mesh, *J Thoracic Surg.*, 26 419 1953
- 18 Brodtkin, H. A., and Peer, L. A. Diced cartilage for chest wall defects, *J Thoracic Surg.*, 28 97 1954
- 19 Heaney, J. P., Cronin, T. D., and Overton, R. C. Unusual problems in management of chest wall defects, *J Thoracic Surg.*, 28 23 1954
- 20 Belsey, R. Civilian chest injuries, *Practitioner* 170 134 1953
- 21 Shefts, L. M. *The Initial Management of Thoracic and Thoraco-abdominal Trauma*, Springfield, Ill. Charles C Thomas, 1956.

# Pleural Emergencies

## 6

### THE MANAGEMENT OF SPONTANEOUS PNEUMOTHORAX

The spontaneous leaking of air from one or both lungs into the pleural cavity is basically a sign of lung disorder the nature of which should, if possible, be determined before it is treated

**Historical Note.** Emerson (1) records that spontaneous pneumothorax was first named by Itard (2) in 1803 and accurately described by Laennec (3) in 1826 After McDowel's (4) report in 1836 of the lesion in a tuberculous patient, the cause was usually accepted as "tuberculous" until Kjaergaard (5) in 1932, analyzing 51 cases, found that spontaneous pneumothorax rarely had anything to do with active tuberculosis, which developed in only one of his series Legget and associates (6) found negative Mantoux tests in 50 per cent of their series This nontuberculous etiology is supported by all subsequent contributors

#### PATHOLOGY

**Etiology.** No age is immune, but healthy males aged 15 to 30 years are most commonly affected Shefts (7), analyzing 114 cases, found the average age was 27 5 years, with the youngest a newborn infant 2 months premature, the oldest 73 years of age. Fifty per cent of the total cases were under 25 years of age and 80 per cent under 30 Males predominate over females in the ratio of 10 1 Harris (8) reported the lesion in 10 newborn infants with respiratory distress.

Rappot (9) divides the causes into five groups

- ✓1. Structural cysts,
- ✓2. Inflammatory disease,
- ✓3. Emphysema, especially the chronic obstructive type,
- ✓4. Trauma, including the postoperative group,
- ✓5. Idiopathic.

The most common cause is rupture of subpleural emphysematous vesicles of two types

- ✓1. A vesicle occurring on localized emphysema without scar tissue,
- ✓2. Multiple apical vesicles beyond the scar of a healed primary tuberculous focus.

Only 6 to 10 per cent of cases of spontaneous pneumothorax are associated with active tuberculosis (9, 10). Rarer causes include rupture of a subpleural staphylococcal lung abscess, especially in infants, partial bronchial occlusion from foreign body or primary or secondary neoplasm (11), ruptured hydatid or giant emphysematous cysts; bronchiectasis, and chest-wall trauma without fractured ribs Spontaneous

## Clinical Features

pneumothorax has also been reported in 36 cases in which it complicated pneumoperitoneum therapy (12) and has occurred on the patient's contralateral side as an unexpected complication of thoracic surgical procedures (13 14) In the author's experience it has also occurred on the ipsilateral side after three days of normal convalescence from ligation of patent ductus arteriosus As D Abreu (15) stresses in some 30 to 40 per cent of patients it develops without obvious signs of pulmonary disease

Regarding the side affected, Crowther (16) reported that, in over 400 cases collected from the literature the relative incidence was 57 per cent on the right side and 43 per cent on the left. It is usually unilateral, but approximately 10 to 12 per cent of spontaneous pneumothoraces are bilateral. In his 71 cases Brock (17) studied 15 bilateral alternating cases and in 8 both sides were involved simultaneously

**Mechanics of Spontaneous Pneumothorax.** A subpleural vesicle may rupture directly into the pleural cavity or air may leak from a deeper bronchiole or alveolus migrate under the visceral pleural surface and form blebs which then rupture into the pleural cavity Occasionally too air treks back along the main bronchus into the lung hilum and mediastinum causing mediastinal and cervical emphysema. When a pleural adhesion is torn, blood or pleural fluid may collect, and if the pneumothorax is due to an infective process an empyema will develop

**Course.** In many cases with an uncomplicated attack, the pneumothorax steadily absorbs until the lung is fully expanded. Provided the pulmonary leak has sealed, the air absorbs at the rate of 1.25 per cent per day (18) the time required being directly proportional to the size of the pneumothorax

Kjaergaard found most absorbed within 6 weeks to 2½ months while Rottenberg and Golden (19) reported the average time required for complete re-expansion was 4 weeks with the shortest 6 days and the longest 12 weeks

But the mere absorption of air is no guarantee against recurrence, for 16 of these same authors 105 cases had 1 recurrence and 8 had 2 or more recurrences with an average time of 23.2 months between attacks Fifteen had recurrence in 1 year or less and 5 in 5 years or more after the first pneumothorax. Brock's (17) 71 cases all had recurrent or chronic spontaneous pneumothorax.

**Complications.** The complications of spontaneous pneumothorax are

- 1 Tension pneumothorax
- 2 Simultaneous bilateral pneumothorax
- 3 Recurrent or chronic pneumothorax
- 4 Pleural effusion
- 5 Hemopneumothorax,
- 6 Empyema.

The first four of these will be discussed in this chapter the last two will be discussed in succeeding chapters

## CLINICAL FEATURES

**Symptoms.** Spontaneous pneumothorax rarely occurs during a period of straining or heavy lifting (10 20) Thirty per cent of Shefts' (7) cases were associated with mild physical activity and in 20 per cent the patients were resting in bed.

The onset is usually sudden and accompanied by a sharp pleuritic pain:

anteriorly or on the shoulder of the collapsed side. Breathlessness and tightness are present. When the lesion is bilateral, respiratory embarrassment and cyanosis are added. The greater the degree of collapse of the lung, the more severe the symptoms.

**Signs. General** Depending on the severity of the pneumothorax, the patient may have little distress, or he may be anxious and apprehensive because of respiratory embarrassment. There may be surgical emphysema of the face, neck, arms, and chest wall.

Further physical signs fall into five groups, of which the first is usual and the rest are complications.

*Pneumothorax Without Tension* The heart and trachea remain central, but there are signs of pneumothorax with unilateral reduced air entry, hyperresonance and diminished breath sounds.

*Bilateral Pneumothorax* When the pneumothorax is bilateral, the mediastinum is again central, but the bilateral hyperresonance and acute respiratory distress point to the diagnosis.

*Tension Pneumothorax* In addition to the signs of a unilateral pneumothorax, there is evidence of increasing tension in the pleural cavity, with displacement of the trachea and apex beat away from the affected side. Physical distress is acute, and, if the intrathoracic pressure is high enough to hamper the function of the normal side, respirations become gasping, the face cyanotic and the pupils enlarged. Finally, if the condition is unrelieved, death ensues from asphyxia.

*Hemopneumothorax* Besides pneumothorax, there are associated signs of blood loss (See Chapter 7.)

*Pyopneumothorax* This is associated with signs of toxemia (See Chapter 9.)

**Differential Diagnosis and Clinical Course.** The sudden pain may occasionally be confused with myocardial ischemia, dissecting aneurysm, spontaneous rupture of the esophagus, perforated peptic ulcer, or cholecystitis with stone.

It is important that all cases of spontaneous pneumothorax be recognized and treated, because if untreated the condition may be fatal, especially if there is tension pneumothorax. This is particularly true in infancy, Harris reported 2 deaths in 10 cases in infants.

**Investigations.** Unless the patient is so distressed that immediate decompression of the pleural cavity is urgently required, there are six important investigations to consider:

- 1 History,
- 2 Chest roentgenograms,
- 3 Sputum examination,
- 4 Bronchoscopy and bronchography,
- 5 Pleural pressure measurement,
- 6 Thoracoscopy

*History* A careful history of previous respiratory disease is most important. In addition to tuberculosis, one should enquire about previous asthma, especially in childhood, and seek evidence of lower respiratory infection including bronchitis and bronchiectasis. Twenty of Brock's (17) cases gave a history of chronic bronchitis, while in an additional 5 bronchiectasis was present.

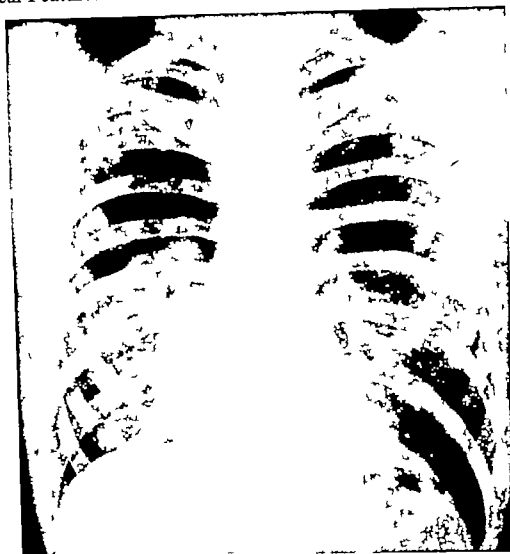


Fig 1 Right spontaneous pneumothorax due to apical bullous cyst.

*Roentgenography* Posteroanterior and lateral films usually disclose

- 1 The extent of the pneumothorax, pulmonary collapse and mediastinal displacement
- 2 The presence or absence of pleural effusion
- 3 The site size and shape of any pleural adhesions
- 4 The probable nature of the underlying lung disease such as cystic emphysema or rarely tuberculosis (Fig 1)

Kircher and Swartzel (18) by calculating on the x ray film the area of two rectangles drawn around the x ray outline of (a) the hemithorax and (b) the collapsed lung, determined the percentage volume of the pneumothorax (Fig 2)

This method not only demonstrates that 1.25 per cent of the pneumothorax is reabsorbed per day but can also be used to estimate length of convalescence if the condition is treated by rest alone

When viewing the plain film it is most important to establish whether the condition is a true pneumothorax or a giant cyst simulating one for if giant cysts are bilateral and are treated with water-seal drains when mistaken for bilateral spontaneous pneumothorax tension pneumothorax will rapidly develop and may even prove fatal

*Tomography* is of value in detecting apical bullas

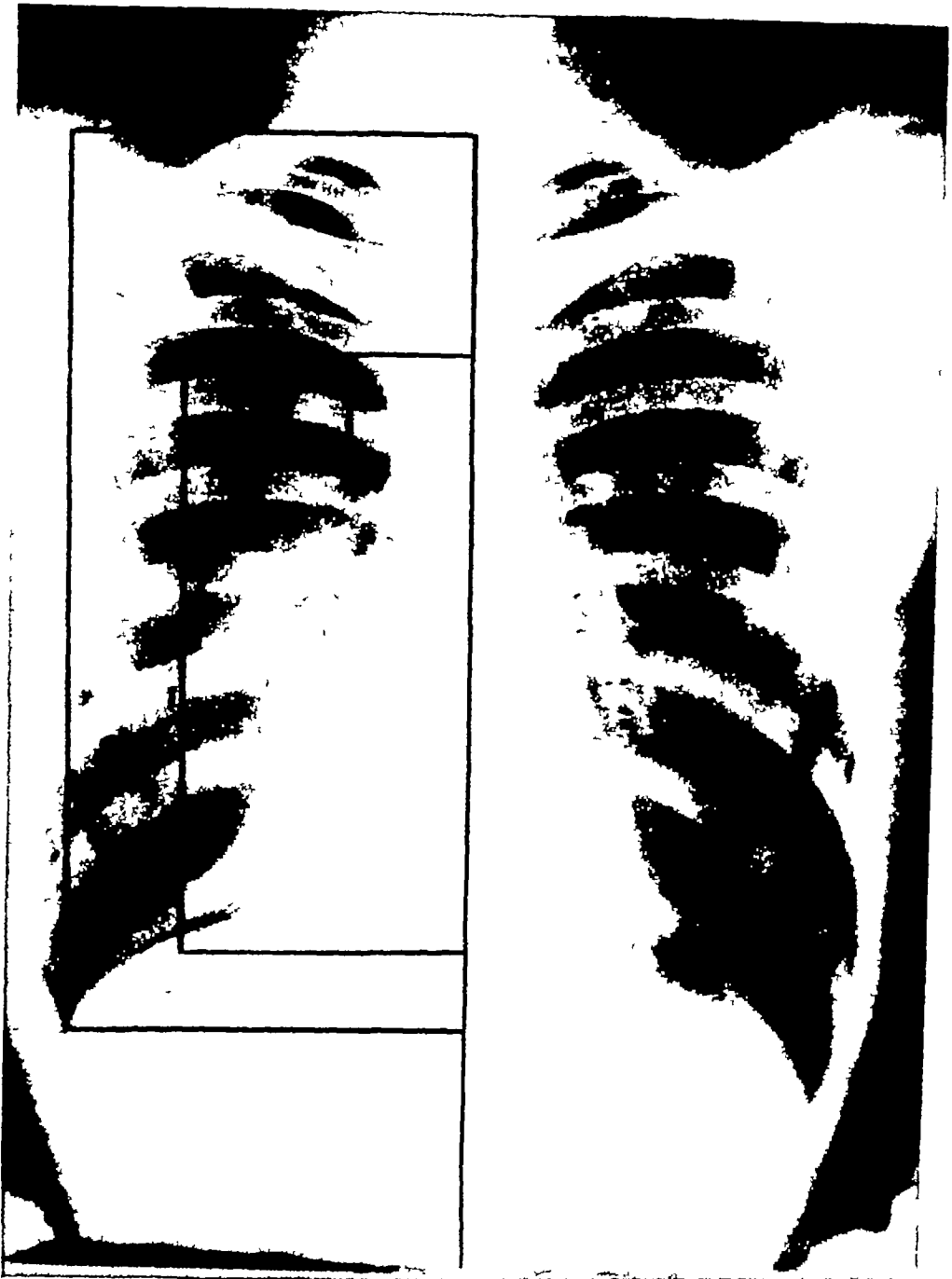


Fig 2 Method of estimating percentage of pneumothorax (Area of pleural cavity minus area of lung gives area of pneumothorax This is expressed as percentage of total area)

*Bronchography* is helpful in (a) detecting unsuspected bronchiectasis, and (b) differentiating between spontaneous pneumothorax and giant bullous cyst.

*Sputum Examination* Sputum if present is examined by direct smear and cultured for organisms, including acid-fast bacilli

*Bronchoscopy* This is at times of great value in disclosing a partial obstruction of the bronchi and should be part of a complete investigation of spontaneous pneumothorax. Figure 5B in Chapter 1 shows a spontaneous pneumothorax caused by a peanut partially occluding the left main bronchus and producing obstructive emphysema beyond. Again, bronchoscopy in a man aged 62 years who had spontaneous pneumothorax revealed an unsuspected neoplasm of the right main bronchus not evident in chest roentgenograms.

## Treatment

**Pleural Pressures** The measuring of intrapleural pressures with a pneumothorax apparatus will show whether or not there is a bronchopleural fistula by the rapid return of the pressure gauge to the initial reading after withdrawal of air. It will also confirm a tension pneumothorax.

**Thoracoscopy** The most valuable and often the most conclusive way of disclosing the actual leak in the lung and its nature is by thoracoscopy, which should never be omitted. It may disclose a ruptured bulla of a bullous emphysema, a polycystic lung, a small bulla beyond an apical scar, or a torn lung. In addition, it is the first step in one well recognized method of treatment.

## TREATMENT

The aims of treatment are

- 1 To secure complete re-expansion of lung as soon as possible,
- 2 To relieve symptoms
- 3 To prevent complications,
- 4 To minimize the period in hospital,
- 5 To secure early return to normal work.

**General Treatment.** With spontaneous pneumothorax considered as a sign of underlying lung disease the foregoing investigations and assessment should be done as soon as possible in order to formulate a sound plan for treatment. There is no place for a policy of *laissez faire*. If the lung does not quickly re-expand of its own accord, active methods can more rapidly and effectively achieve the desired result.

**Thoracentesis Alone.** This is of doubtful value and is not advised. Rapport (9) states that of five patients treated by this method only one recovered uneventfully at the end of six weeks.

**Intrapleural Needle and Water-Seal Drainage.** Basically this is of value as an emergency measure only and should never be relied on as the sole definitive method of treatment. For example in two cases it was used when the patients were transported 80 and 100 miles respectively to a thoracic surgical unit.

In one case—as there was no closure after three weeks and as the patient's condition was deteriorating and he was developing auricular fibrillation—thoracotomy, excision of the ruptured bulla, and lung suture were advised, these procedures gave a rapid sure cure.

The second case illustrates the pitfalls of the method and the fallacy of a *laissez faire* policy. The patient was admitted to the medical ward after a 100-mile journey with a needle and water-seal drainage controlling the spontaneous pneumothorax. Within 24 hours the needle had broken and the point lay in the chest wall, where it could not be located. The patient was treated expectantly one month later the pneumothorax recurred. When he was finally referred, the needle lay deep in a cystic left upper lobe. Thoracotomy allowed removal of the needle, closure of the leak, and induction of artificial pleurisy to prevent recurrence. (See Chapter 13, Fig. 7.)

**Intercostal Tube with Water-Seal Drainage.** Where thoracic surgical facilities are available this is not advised as definitive treatment. It does not strike at the heart of the problem—the leak from the lung—nor does it provide any assurance against recurrence. Like an intrapleural needle, it should be regarded as an important emergency method, especially with tension or bilateral pneumothorax.



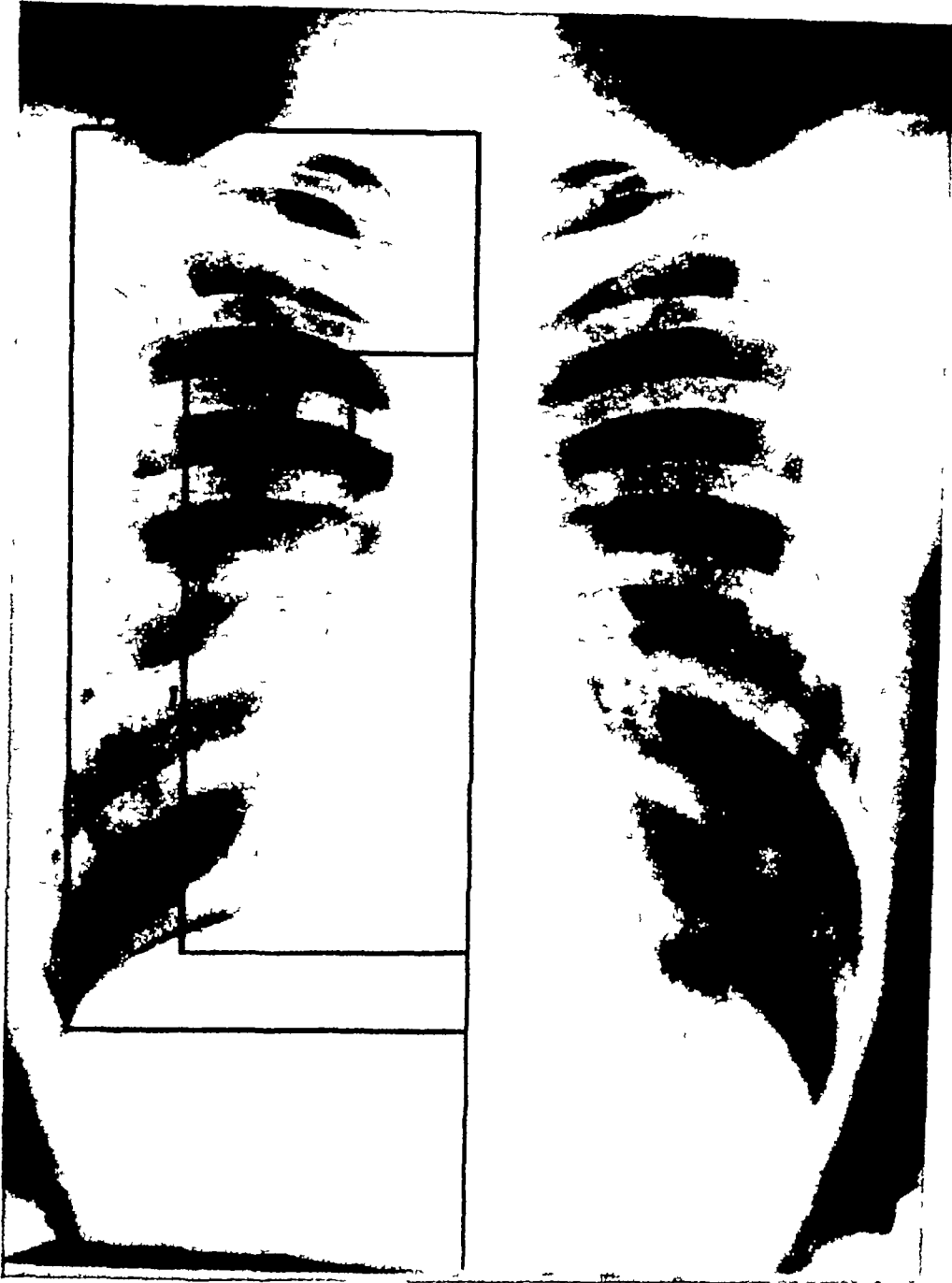


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## PROBLEMS ASSOCIATED WITH SPONTANEOUS PNEUMOTHORAX

The problems of spontaneous pneumothorax may present themselves in seven different ways, each requiring variation in management:

- 1 Tension pneumothorax,
- 2 Pneumothorax simplex of less than 10 per cent,
- 3 Pneumothorax simplex of more than 10 per cent,
- 4 Recurrent pneumothorax and that due to obvious lung lesions,
- 5 Pneumothorax from intrabronchial foreign body,
- 6 Resistant pneumothorax,
- 7 Postpneumectomy spontaneous pneumothorax

**Tension Pneumothorax.** This is a true emergency, requiring prompt recognition and treatment

In the most dire case, a stab in the chest wall with a scalpel will allow escape of air and the more leisurely insertion of a water-seal drain

Alternatively, a needle inserted into the pleural cavity and attached by a tube to water-seal drainage usually functions for 24 hours. With bilateral pneumothorax this treatment is of even greater value as a first-aid measure. However, as already stressed, this method neither removes the cause nor guards against recurrence, and an artificial pleurisy must later be induced (See below)

**Pneumothorax Simplex of Less Than 10 Per Cent.** If serial roentgenograms of a first attack show a steady absorption of air and re-expansion of the lung during the first week, the pneumothorax can be treated conservatively by rest. The principal disadvantage is that it takes a month or more to re-expand the lung (19). In addition, both the patient and the physician should understand that recurrence can occur in 30 to 40 per cent of cases (16, 21) and that this method is no safeguard against recurrence which can be more severe and most inconvenient when the patient is far from medical aid. The only true security for the future lies in obliterating the pleural cavity

**Pneumothorax Simplex of More Than 10 Per Cent.** As this disease occurs mainly in fit, young, family-supporting males, the prospect of treatment by bed rest alone, with its prolonged convalescence and no security for the future, is too unrealistic. In a first attack, when there is no x-ray evidence of a bullous cyst or serious lesion, the most satisfactory approach is as follows (Fig 3A)

- 1 The patient is well sedated with Morphine 22 mg and scopolomine 0.43 mg, and under local anesthesia a thoracoscope is passed in the midaxillary line.
2. The apex of the lung is inspected and, as likely as not, a small apical bleb will be seen. If there is an adhesion, this is divided by electrocautery.
3. An artificial pleurisy is next induced by liberally puffing iodized talcum powder throughout the pleural cavity. The author has used this method over the past eight years and has found that it gives an excellent pleural reaction without the severe pain and excessive fluid formation of silver nitrate solution. Further, the wide coverage ensures a generalized pleurisy as opposed to the localized reaction from injecting camphorated oil (which is more valuable as a counterirritant *on* the chest rather than *in* it).
- 4 To ensure immediate and total re-expansion of the lung, a Jacques or Malecot rubber catheter is next introduced through the thoracoscopy cannula and connected to water-seal drainage and continuous suction (Fig 3B).



Fig. 3A. Total left spontaneous pneumothorax.

- 5 The patient is returned to the ward via the x ray department, where films confirm the re-expansion of the lung. Continuous suction is maintained at a pressure sufficient to remove all escaping air usually 5 to 10 mm. Hg. If the lung has not re-expanded, bronchoscopy is required, lest there be retained endobronchial secretion requiring aspiration (22)
- 6 The fistula usually closes within 36 hours and the tube becomes blocked within 48 hours. After a further roentgenographic check of full lung expansion, the tube is removed.
- 7 If an apical pocket of air persists this is aspirated in the first instance but if the fistula is still present, the intercostal tube is reinserted higher up and a further 48 hours of suction and roentgenographic check carried out.

**CONVALESCENCE** The patient has pain for two or three days thereafter it lessens, and he can usually be discharged within a week and be fit for light duties within two weeks. With adequate treatment and careful tube management, recurrence is unknown (Fig 3C)



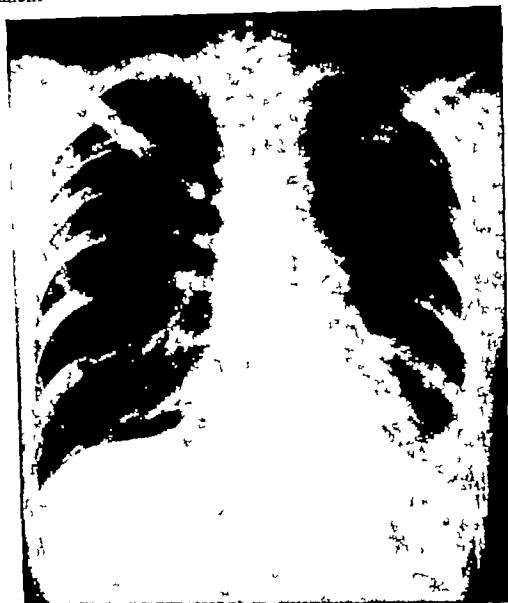


Fig. 3C. Fistula closed and lung well aerated one week later

*Bronchopleural-cutaneous fistula* is more likely to occur (a) when no suction has been used or (b) when there is a large apical emphysematous cyst or cysts. Thoracotomy is advised, and this procedure can allow excision of the causative lesion. If still persistent, then thoracoplasty is advised (Fig. 4D)

**Pneumothorax That Is Recurrent or Due to Obvious Lung Lesions.** Here the same small apical blebs as mentioned above, large or giant cysts or a polycystic lobe are the usual causes. In particular the last named require prophylactic resection before they rupture and produce tension pneumothorax. On other occasions a pulmonary neoplasm or localized bronchiectasis is discovered at investigation.

These cases require thoracotomy and pulmonary resection, be it wedge resection, segmental resection, lobectomy or pneumonectomy according to the lesion being treated. After resection is complete iodized talcum powder is blown over any remaining lung tissue and the chest wall is closed over water-seal drainage. Convalescence is usually rapid, and most patients are discharged within 14 days. Ehrenhaft and associates (23) reported thoracotomy and resection in 19 cases, with eradication of the lesion and prompt and complete re-expansion of the lung.

**Pneumothorax from Intrabronchial Foreign Body** (see Chapter 1, Fig 5B). Treatment is primarily endoscopic removal of the foreign body. In the author's experience, this has sufficed. If the pneumothorax does not absorb with rest alone, aspiration and thoracoscopy may be required.

**Resistant Pneumothorax.** Spontaneous pneumothorax occurring in a patient with advanced, generalized emphysema may prove most resistant to treatment. The following case report illustrates the complexities of management (Fig 4).

**CASE REPORT.** R. J., aged 52 years, was admitted to a country hospital on March 17, 1955, with severe left spontaneous pneumothorax. Initial treatment consisted of thrice-daily aspiration of air from the left pleural cavity. As this was ineffective, on the tenth day of his illness he was admitted to Dunedin Hospital. The admission roentgenogram showed apical scarring, generalized pulmonary emphysema, surgical emphysema of the chest wall, and complete collapse of the left lung (Fig 4A).

As the patient was in poor physical condition, with low respiratory reserve and blood pressure of 104/80 mm Hg, immediate intercostal water-seal drainage was instituted. It relieved the tension but did not close the fistula (Fig 4B).

On the twelfth day, he had thoracoscopy, artificial pleurisy with iodized talcum powder, drainage, and continuous suction (Fig 4C). On the twenty-fifth day as there was still a large persisting fistula, left thoracotomy was performed through the fifth rib-bed. The pleural layers were partially adherent, but at the apex was a ruptured giant bullous cyst communicating with a system of bullous cysts. The tear was oversewn, and tension on the suture line was relieved by stripping the upper lobe from the parietes in the extrapleural plane. In order to produce a more severe pleurisy, the lung was painted with 10 per cent silver nitrate solution. The chest wall was closed in layers with catgut over water-seal drainage. Continuous suction was again instituted.

On the fifty-fifth day, as the fistulas still persisted and as the space was infected, the drainage was made more dependent by rib resection, on the sixty-ninth day of his illness, an apical three-rib thoracoplasty was performed. The extrapleural space required postoperative drainage, and the patient was discharged fit for work on September 17, i.e. 153 days after onset (Fig 4D).

In summary, therefore, his treatment was:

- 1 Aspiration,
- 2 Intercostal water-seal drainage;
- 3 Thoracoscopy—poudrage—drainage and suction,
- 4 Drainage of apical empyema;
- 5 Thoracoplasty with drainage.

*Comment.* The extreme degree of apical scarring and bullous cyst formation on an old tuberculous infection made for complicated, protracted treatment in this patient.



Fig. 4A. Resistant spontaneous pneumothorax. Left tension pneumothorax, admission film.



Fig. 4B. Film after intercostal water-seal drainage.





Fig 4C After thoracoscopy, poudrage and continuous suction, persisting fistulas

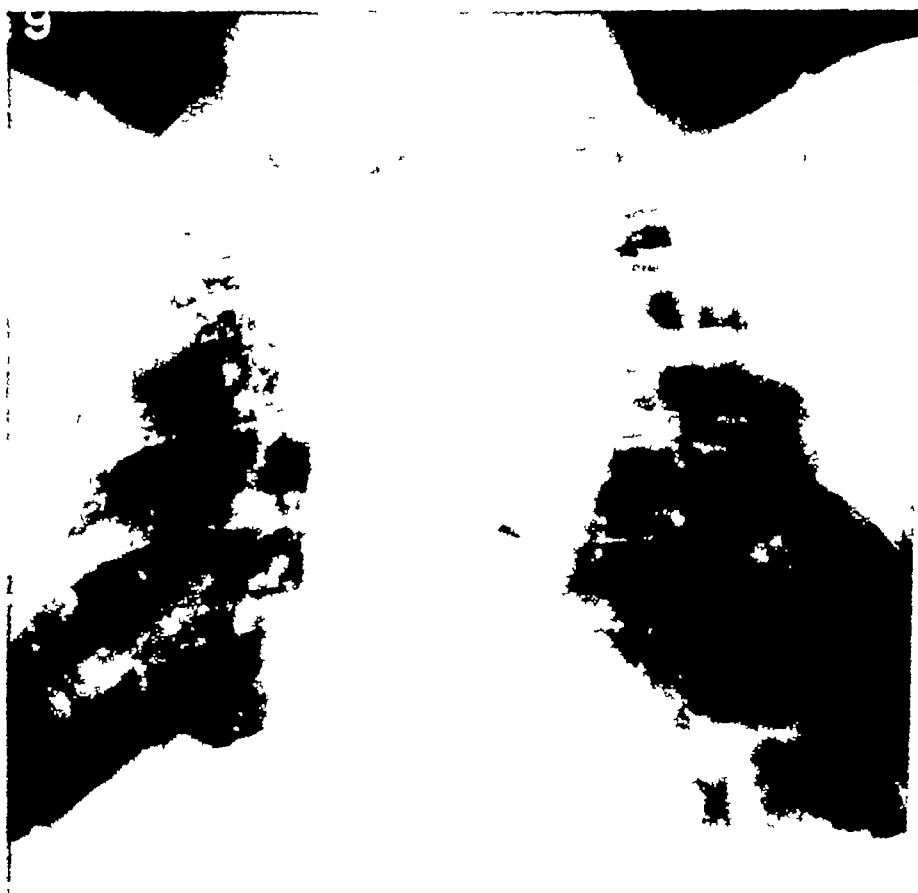


Fig 4D Final closure by three rib apric il thor icoplasty



Fig. 5A. Right spontaneous pneumothorax after left pneumonectomy

**Postpneumonectomy Spontaneous Pneumothorax.** In these days of routinely successful pneumonectomy emphysema in the remaining lung is almost inevitable. Rarely a postoperative spontaneous pneumothorax will develop with serious consequences. Management is as follows:

- 1 Chest roentgenogram to confirm the diagnosis
- 2 Intercostal water-seal drainage to make the patient safe
- 3 Induction of an artificial pleurisy

**CASE REPORT (Fig. 5)** R. C., a lad aged 10 years, had left pneumonectomy in September 1954 for total left bronchiectasis. Three months later—while on a carousel at a seaside show—he became “tight in the chest” and was taken to the hospital. A right pneumothorax was aspirated, and he traveled 140 miles home. Two weeks later the pneumothorax recurred, and he was admitted in a distressed state to the pediatric department where an intercostal needle was inserted. This did not seal the leaks, and on January 1, 1955 he was referred for induction of an artificial pleurisy.

With the patient intubated to allow of controlled anaesthesia, a trocar and cannula were inserted into the right pleural cavity and poudrage with iodized talc was performed. Two Malecot catheters were inserted—one as a drain and one as an air vent—and connected to water seals and suction.

On January 31 10 oz. of straw-colored fluid were aspirated; thereafter convalescence progressed satisfactorily.



Fig 5B Right lung re-expanded after thoracoscopy, poudrage, and suction drainage



Fig 5C Chest film eight weeks later

## CONCLUSIONS

Spontaneous pneumothorax is an emergency that requires prompt adequate treatment, if prolonged convalescence and recurrences are to be avoided. All cases of recurrent spontaneous pneumothorax, chronic pneumothorax and bilateral recurrent pneumothorax require thoracotomy

## REFERENCES

- 1 Emerson, C. P. Pneumothorax a historical, clinical and experimental study Johns Hopkins Hosp Reports 11 1 1903
- 2 Itard, J. E. M. G. Sur le pneumothorax, ou les congestions gazeuses qui se forment dans la poitrine, Paris, 1803
- 3 Laennec, R. T. H. Traité de l'Auscultation Médiate et des Maladies des Poumons et du Cœur 2nd ed., Paris, Chande, 1826. 2V., XXXVI, 728 pp. 4 pl. 790 pp., 11 pl.
- 4 McDowell, On an unusual form of pneumothorax, Dublin Hosp Gaz., 3 227 1856
- 5 Kjaergaard, H. Spontaneous pneumothorax in the apparently healthy Acta. med. Scand. Supp. 43 1932.
- 6 Leggett, E. A. Myers, J. A., and Levine, I. Spontaneous pneumothorax, report of 31 cases, Am. Rev. Tuberc., 29 348 1934
- 7 Shefts, L. M. Gilpatrick, C., Swindell, H., and Gabbard, J. G. Management of spontaneous pneumothorax, Dis. of Chest, 26 273 1954
- 8 Harris L. E. Pneumothorax in the newborn infant, Proc. Mayo Clinic, 30 297 1955
- 9 Rapport, R. L. Thurlow A. A. and Klamm, K. P. Etiology and management of spontaneous pneumothorax, A.M.A. Arch. Surg., 67 266, 1953
- 10 Briggs, J. N., Walters, R. W., and Bryon, F. X. Spontaneous pneumothorax, Dis. of Chest, 24 564 1953
- 11 Shaw A. B. Spontaneous pneumothorax from secondary sarcoma of lung, Brit. M. J. 1 278 1951
- 12 Shapiro M. Spontaneous pneumothorax complicating pneumoperitoneum therapy Ann. Intern. Med., 43 876 1955
- 13 Beno, T. J. and Welsel, W. Spontaneous contralateral pneumothorax complicating thoracic surgical procedures, J. Thoracic Surg., 23 272 1952.
- 14 Gleason, G. E. and Kent, E. M. Contralateral spontaneous pneumothorax following lobectomy J. Thoracic Surg., 18 473 1949
- 15 D'Almeida, A. L. A practice of Thoracic Surgery London, Edward Arnold & Co., 1953
- 16 Crowther J. S. Spontaneous pneumothorax, a review of 61 cases, Tubercle, 36 265 1955
- 17 Brock, R. C. Recurrent and chronic spontaneous pneumothorax, Thorax, 3 88 1948
- 18 Kircher L. T., and Swartzel, R. L. Spontaneous pneumothorax and its treatment, J.A.M.A., 155 24 1954
- 19 Rottenberg, L. A. and Golden, R. Spontaneous pneumothorax A study of 105 cases, Radiology 53 157 1949
- 20 Marrangoni, A. G. Storey C. F. and Gelb P. O. The management of spontaneous pneumothorax, Am. Rev. Tuberc., 72 257 1955
- 21 Myerson, R. M. Spontaneous pneumothorax in a clinical study of 100 consecutive cases, New Eng. J. Med., 238 461 1948
- 22 Rubin, M. and Rubin, E. H. Bronchoscopy in the treatment of spontaneous and traumatic pneumothorax, J. Thoracic Surg., 21 377 1951
- 23 Ehrenhaft, J. L., Taber R. E., and Lawrence, M. S. Spontaneous pneumothorax A review with the results of pulmonary resection in 19 patients, Am. Rev. Tuberc., 72 861 1955

## SPONTANEOUS HEMOPNEUMOTHORAX AND HEMOTHORAX

**Introduction.** Spontaneous hemopneumothorax, the most serious complication of spontaneous pneumothorax, requires fuller consideration. In 1828 Laennec (1) referred to an autopsy in which "upon penetrating the left side of the chest, a large quantity of inodorous gas made its escape with a hissing sound" from a pleural cavity that contained "about ten ounces of a bloody serosity." In 1876, aspiration was successfully used by Whittaker (2) whose patient, after the removal of 34 oz. of bloody fluid and air from his chest, made an uncomplicated recovery.

In 1900, both Pitt (3) and Rolleston (4) each independently reported a fatal case in a young man, and Pitt gave the lesion the present-day name of "spontaneous hemopneumothorax." In 1948, Elrod and Murphy (5) first published an account of pulmonary decortication for this lesion, while in 1951 Neuhof (6) recorded having done such an operation in 1936.

In 1951, Myers and associates (7) described the first "early" thoracotomy 60 hours after onset of symptoms. Ross (8) and the author (9), in 1952 and 1953, each reported successes with emergency thoracotomy for massive spontaneous hemopneumothorax, and since then this direct approach to secure the bleeding point has become a standard procedure. In 1955, Fry and associates (10) found a total of 174 cases in the literature, and they published an extensive bibliography.

### PATHOLOGY

Both spontaneous hemothorax and hemopneumothorax are complications of spontaneous pneumothorax. The lesion is 15 times as common in males as in females (11). The ages most commonly affected are between 17 and 32 years, and the average age incidence for all reported cases is 28 years (10). The right and left sides are affected equally. Though unusual, recurrence has been authenticated (12), while an earlier or later spontaneous pneumothorax has been described (13, 14).

**Pathogenesis.** Hemothorax may be expected in 8 to 10 per cent of patients with spontaneous pneumothorax. The bleeding may arise in two ways, either 1, from a vessel in the wall of the torn cyst, or 2, from mounting intrapleural tension stretching and finally rupturing an adhesion. If the fistula persists and has a "ball valve" mechanism, a tension hemopneumothorax results. Helwig and Schmidt (15), reporting 14 fatal cases examined at autopsy, found 4 with torn adhesions, 4 with ruptured blebs and 6 in which no bleeding point was detected.

**Course.** The bleeding is insidious and steady, and it is clinically latent until 1 to 1.5 liters have been lost. The pleural cavity steadily fills with blood which rapidly clots and becomes defibrinated. As much as a 10-liter bleed has been recorded (10).

Rarely, especially when the patient has a blood dyscrasia, spontaneous hemothorax results with similar problems (16, 17).

Depending on the severity of the bleeding, there are two effects.

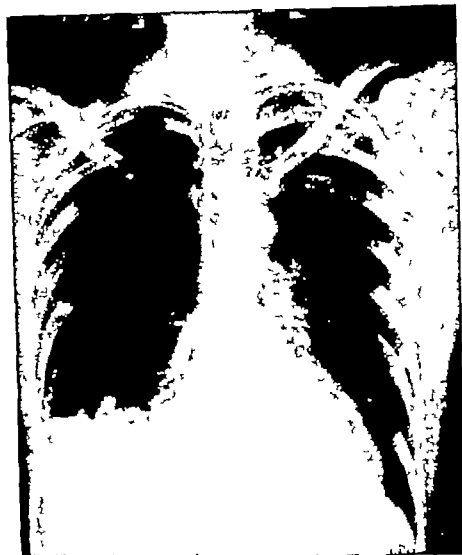


Fig 1A. Chest film showing small right spontaneous pneumothorax (Feb 26 1952 5 30 P.M.)

- 1 *Locally* the lung is collapsed by the hemothorax and is held collapsed by fibrin which is deposited in thick layers on the parietal and visceral pleurae thus seriously restricting lung function
2. *Systemically* there are the effects of blood loss. Massive bleeding, unrelieved, is rapidly fatal within 24 to 48 hours

Cosgriff (18) has clearly shown that the hemothorax clots early and is followed by the usual phenomenon of fibrinolysis and by the deposition of fibrin on the collapsed lung

### CLINICAL FEATURES

The onset may be sudden or insidious and occur while the patient is resting. Occasionally it occurs during exercise (9)

Typically the patient, a healthy young male, suddenly develops spontaneous pneumothorax with accompanying tightness in the chest, shortness of breath, and pain which may radiate to the neck, shoulder or upper abdomen. The symptoms may abate, but usually they recur within 24 to 48 hours with faintness, sweating, shock, collapse, a rising pulse rate and falling blood pressure indicating blood loss. In ad-

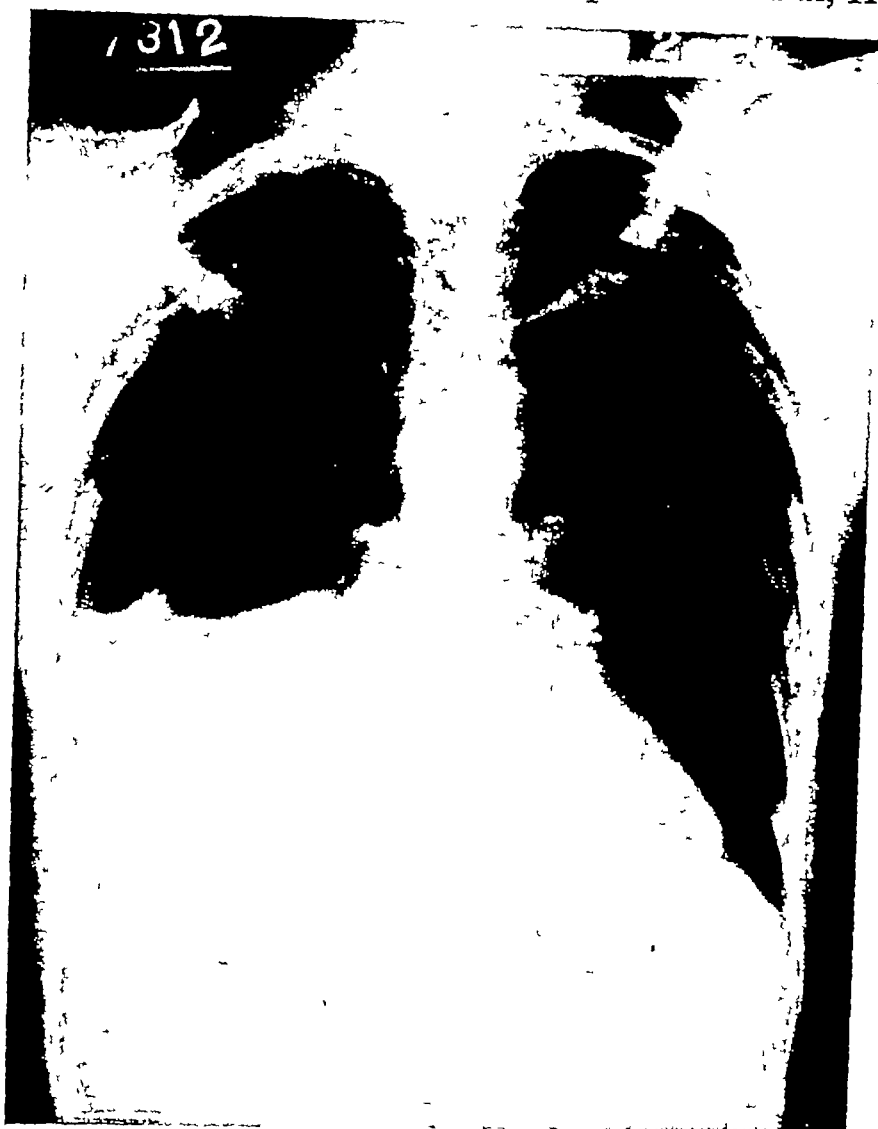


Fig 1B Same day (7 30 P M ), rapid increase in blood loss, with total collapse of right lung  
Emergency thoracotomy Bleeding from wall of torn apical bullous cyst

dition, physical examination of the chest shows the classical signs of pleural effusion and perhaps displacement of the mediastinum to the opposite side

If the patient is first seen some days after hemorrhage has ceased, there will again be signs of pleural effusion as well as anemia. There may be slight pyrexia, leukocytosis, and a raised erythrocyte sedimentation rate.

**Investigations.** Awareness is the way to prompt diagnosis and treatment. The four important investigations are.

1. *Clinical Examination* This will reveal signs of air and fluid in the pleural cavity, and of anemia,
2. *Roentgenography.* Anteroposterior and lateral films support the clinical findings. When repeated at hourly or two-hourly intervals, they show if bleeding is continuing and if the mechanics of the lung are being altered (Figs 1A-C)
3. *Chest Aspiration* This confirms the diagnosis and *must never be omitted*. The aspirate may be fluid; but if clotted, only a few flecks of blood clot will be drawn into the syringe.
4. *Blood Examination* Blood grouping is essential. Serial hemoglobin and packed-cell volume estimations indicate the rate and extent of blood loss and what volume must be replaced.

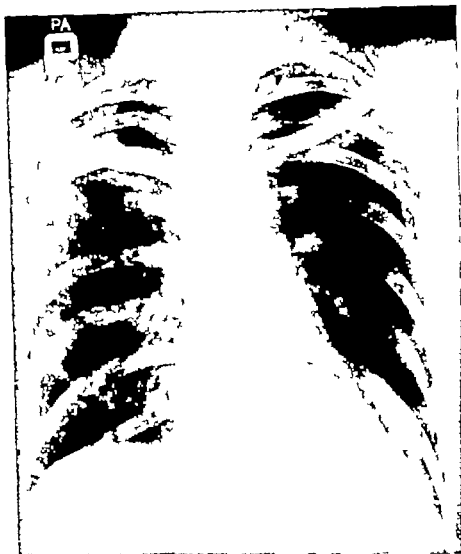


Fig. 1C. Chest film taken 10 days after operation (Mar 7 1952)

**Differential Diagnosis.** Hemopneumothorax can readily be mistaken for other lesions especially if the pleural signs have not been detected. Calvert and Smith (11) report the recorded errors in diagnosis. Irritation of the diaphragm may produce signs simulating an acute abdominal emergency while nausea, vomiting and even abdominal rigidity may suggest a bleeding or perforated peptic ulcer acute pancreatitis or appendicitis as well as biliary or renal colic. Again, the pain may suggest coronary thrombosis or in the elderly a dissecting aneurysm.

**Prognosis.** Most deaths occur within two days of onset, from exsanguination. Whereas the mortality of reported cases in 1951 was 25 per cent, by 1955 it had fallen to 12 per cent. Even this figure may be high, because mild cases have not been recorded. Nevertheless it indicates the seriousness of this lesion which is essentially "internal hemorrhage into a cavity capable of holding the circulating blood volume."

As Clyne and Hutter (19) point out, "reluctance to operate has led to the high mortality with a chest disability in survivors which at the best results in slow convalescence with probably some permanent pleural thickening while less fortunate cases will require the major procedure of pulmonary decortication to correct a crippling chest condition. One must remember that after initial resuscitation the patient, a healthy young adult, is fitter for surgery than he is likely to be for a long time if surgery is delayed, and that the real risk in these cases lies not in operating, but in not operating."



**TREATMENT**

*The objects of treatment, therefore, are to*

- 1 Resuscitate the patient and replace blood loss;
- 2 Arrest the bleeding and prevent its recurrence,
3. Remove all air, blood, and clot from the pleural cavity;
4. Obtain and maintain full lung expansion, so that there is a return to full normal function in a minimum of time

Management depends on whether the patient is seen early or late.

**THE ACUTE STAGE**

Whether the condition is mild or severe, the following routine is advised.

1. **Sedative.** Morphine 15 mg. or pethedine 100 mg will relieve the patient's immediate anxiety
2. **Blood Transfusion.** Even if the hemothorax is small, packed-cell volume, hemoglobin, and blood grouping must be done as a safeguard against further bleeding. If serial roentgenograms show bleeding is continuing, blood loss must be replaced by transfusion. In one of the author's cases (9) the patient required 4.5 liters of blood in six hours, given simultaneously into an arm and a leg vein
3. **Intercostal Drainage.** When there is an associated tension pneumothorax, temporary intercostal drainage is lifesaving, but, because of difficulty in removing the associated blood clot, intercostal drainage cannot be advised as a permanent measure. As Ross (8) advised, one should be prepared to do a thoracotomy if necessary
4. **Aspiration.** The diagnosis is usually confirmed by aspirating the chest through the eighth or ninth intercostal space below the angle of the scapula. At best, further aspiration by removing most of the blood and air will allow the lung to re-expand and the pleural surfaces to adhere. A chest film is thereafter taken. If the lung has re-expanded, it may be watched by serial roentgenograms at eight-hourly intervals. If, however (as is more common) there is no alteration in the shadow, or if the bleeding continues, other treatment is required
5. **Fibrinolytic Enzymes.** Fibrinolytic enzymes have a limited place in treating a small hemothorax with arrested bleeding. Though Read and Berry (20) have reported successes, the author has found their use disappointing. Beside the initial reaction, the patient must withstand repeated thoracenteses with the distinct possibility that the thick fibrinous walls coating the lung will be unaffected by the lytic process. In addition, intrapleural infection may lead to chronic empyema and the prospect of decortication or an unroofing operation. Repeated use of these enzymes has also led to anaphylaxis (21). They offer no security against further bleeding and may even cause it to recur by dissolving the blood clot over a bleeding vessel. Re-expansion of the lung is not guaranteed, nor does the method approach the ease and safety of thoracotomy.

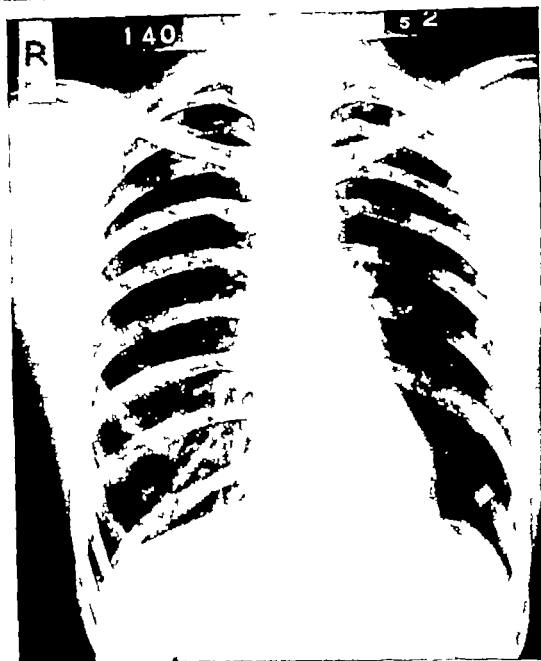


Fig. 2A. Right spontaneous pneumothorax (Jan. 11 1952)

### THORACOTOMY

In most cases the only way to fulfill the aims of treatment, reduce morbidity and secure prompt return to full useful employment is by thoracotomy. This is of especial value when the bleeding has been sudden massive and is continuing. Without operation such cases are rapidly fatal. The correct attitude to adopt is "Now is the hour" (8) (Figs 2A-C.)

**Preoperative Management.** The patient is resuscitated, the blood pressure improved to over 100 mm Hg, and the hemoglobin raised to at least 80 per cent of normal with one or more blood drips. He is then prepared for thoracotomy with the usual skin toilet. The only premedication required is atropine 0.65 mg.

**Anesthesia.** Because of the blood clot, and air collapsing the affected lung and because of the distinct possibility of pleural tension phenomena a cuffed endotracheal tube is essential. A further safeguard is to insert temporarily an intercostal drainage tube into the pleural cavity and so relieve any tension pneumothorax. There

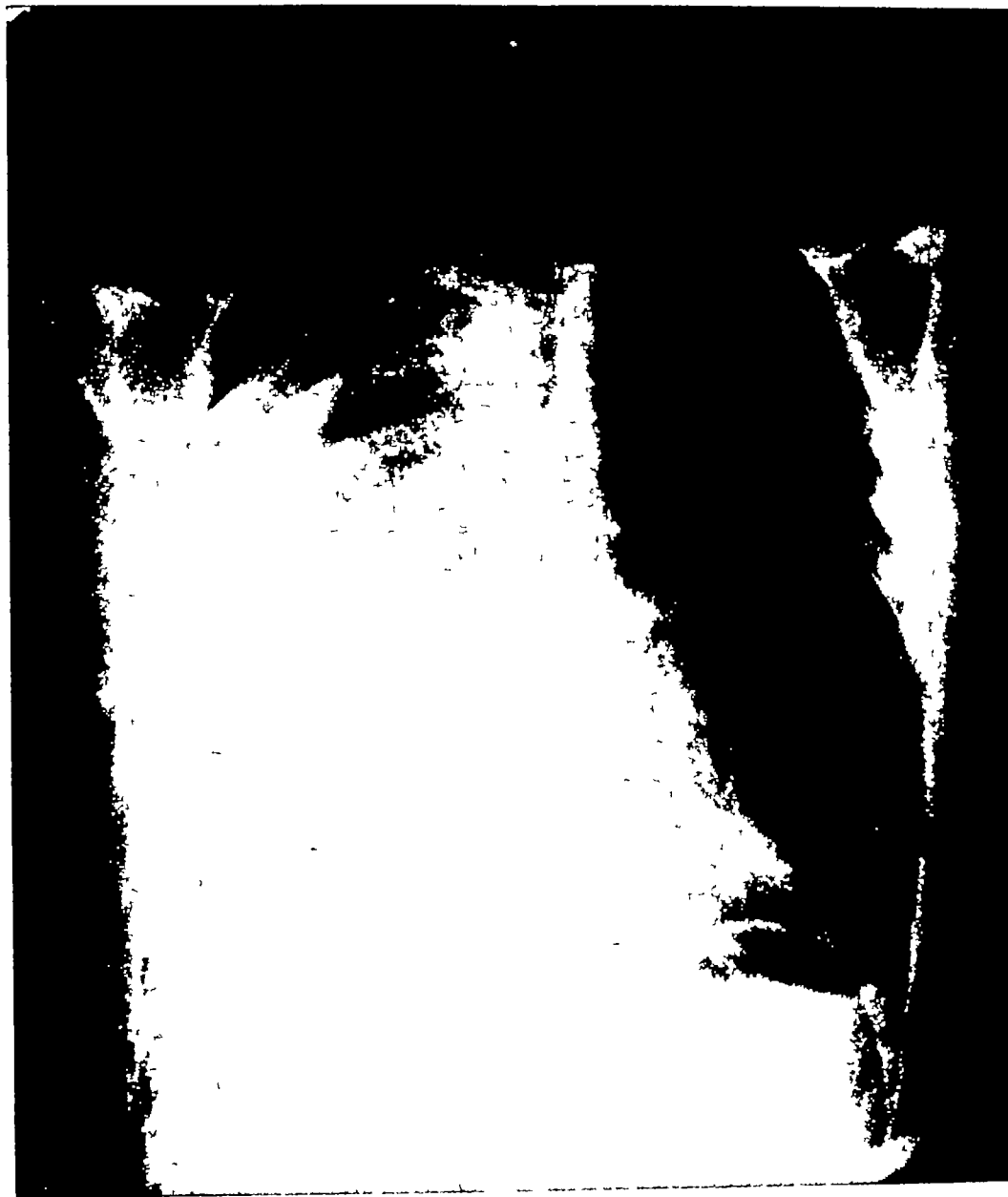


Fig 2B Massive hemothorax, emergency thoracotomy, bleeding from adhesive stump on parietal pleura (Jan 13, 1952)

after, light anesthesia and oxygen suffice. The surgeon also infiltrates the thoracotomy site with a local anesthetic solution.

**The Operation.** The chest is opened through a standard sixth-rib-bed approach. When the rib is resected, the underlying pleura has a characteristic black color. After the pleura is opened, the blood clot, as much as 2.5 liters, is manually removed, the pleural cavity sucked clear, the lung freed and then re-inflated by the anesthetist. A search is made at the apex of the lung and in the pleural cavity for the bleeding point.

In two of the author's cases treated by emergency thoracotomy, the bleeding point in one lay at the parietal pleural end of a torn apical adhesion, in the other it was from the wall of a torn apical bullous cyst. The first case was arrested by touching it with continued suction in connection with diathermy. The second case required wedge resection of the oozing, ruptured, cystic area by clamping with Maingot's central ridged pedicle forceps and oversewing in two layers with 0 chromic catgut mounted on an eyeless needle.



Fig 2C. Postoperative re-expansion of right lung (Jan. 14 1952)

An artificial pleuritis is next established with talc and iodine powder or by painting the parietes with 3 per cent silver nitrate solution. The lung is fully re-expanded and the chest wall closed in layers over two water seal drains—one as a drain and the other as an air vent. Throughout the procedure blood loss must be replaced

**Postoperative Management.** On return to the ward, the patient requires

- 1 Continuous intrapleural suction to keep the lung fully expanded
- 2 Sedatives such as morphine 15 mg or pethidine 100 mg six-hourly
- 3 Antibiotic therapy
- 4 Iron therapy such as colliron 4 ml. thrice daily

The patient lies on the operated side with head down and the foot of the bed raised, in order to encourage blood clot to cover and seal any residual lung leak.

The tubes will usually cease draining air or fluid within 24 hours. If a chest roentgenogram confirms that the lung is fully expanded the intercostal tubes are re-

moved. If, on the other hand, there is still a pneumothorax, a fresh intercostal drain must be inserted at the required site and the earlier ones removed. Atelectasis is treated by bronchoscopic aspiration with further check roentgenograms.

In practice, these patients do well. The lung is re-expanded at operation and remains re-expanded thereafter. Sutures are removed on the seventh or eighth day, and the patients can be discharged between the tenth and fourteenth days. Convalescence is minimal, and return of full lung function and normal activity is maximal.

### THE CHRONIC STAGE

**Object of Treatment.** When seen in this stage, the bleeding has ceased, the fistula has usually closed, and the patient is partially adjusted to his blood loss, collapsed lung, and mediastinal displacement. The aim of treatment is early and complete re-expansion of the lung that is held collapsed by layers of fibrin and fibrous tissue. There are two methods:

- 1 *Chest Aspiration.* In early cases, repeated and complete chest aspiration has been reported to be of value,
- 2 *Decortication.* In the more common established case, however, with a large hemothorax, the only adequate way to secure these aims is by decortication, to remove the encasing fibrin.

**Preoperative Treatment.** Chest roentgenograms and blood examination are necessary, while differential bronchspirometry shows how limited is the ability of the affected lung to absorb oxygen. Bronchoscopy confirms that there is no endobronchial lesion to hinder complete re-expansion of the lung after decortication.

**The Operation.** Depending on the site and size of the chronic hemothorax, the chest is opened through the bed of the sixth to eighth ribs. In large decortications, it may be necessary to resect the sixth rib to approach the upper pleural cavity and the eighth to ninth ribs to free the diaphragm. On opening the pleural cavity, a dense wall of fibrous tissue is found. This is stripped digitally from the parietes, and a rib retractor is inserted.

Thereafter, it is wise to commence from below and in front, dissecting free the diaphragm, the lower lobe, the phrenic nerve, and finally working back toward the aorta. As the lung is released, it re-expands and fills the pleural cavity. After the major "peel" has been removed, further constricting fascial bands are separated from the lung surface.

After securing hemostasis with diathermy, gelatin sponge and similar means, the chest wall is closed over two water-seal drains which are connected to continuous high-pressure suction on return to the ward.

**Postoperative Management.** The routine measures have already been mentioned. The major objective is to keep the lung re-expanded and the visceral and pleural surfaces adherent so that parenchymal fistulas will close. This requires continuous suction (see Chapter 2), and daily roentgenographic check of the lung. The complications—sputum retention and pleural infection—are discussed in Chapter 12. Supportive antibiotic and iron therapy—colliron 4 ml thrice daily—assist correction of the anemia.

Postoperative check bronchspirometry will reveal if the lung does in fact absorb oxygen or is just a space-occupying organ, obliterating the pleural cavity. The complexity of management of such cases is illustrated in the following case report (Figs 3A-B).

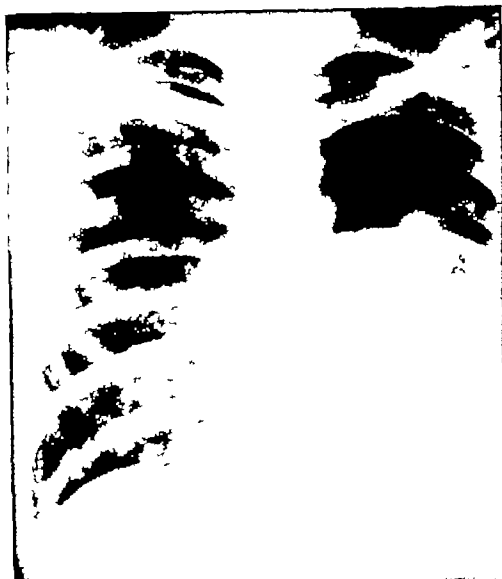


Fig. 3A. Chest film (made June 23 1954) five days after onset of left hemopneumothorax, showing adhesions and collapsed left lung.

CASE REPORT D. A. a man aged 34 years, was seated at breakfast on June 18 1954 when he experienced left chest pain which rapidly increased in severity and spread to the left shoulder. On June 21 he was admitted, cyanosed and breathless, to a country hospital. His pulse was 140 per minute, respiration 30 per minute and he had signs of a left tension pneumothorax with the trachea and heart displaced to the right of the sternum. He was considered to have a spontaneous hemopneumothorax which was treated by thoracentesis with great relief. The needle was left in situ and attached to a water-seal drain for three hours after which it became blocked and was removed.

On June 23 he had 1.2 liters of blood transfused and was transferred 100 miles to Dunedin Hospital. On June 24 450 ml. fluid were aspirated and the hemoglobin was 9 gm. per 100 ml. His temperature rose to 102° F. As there was no great radiologic improvement, as expectant treatment would prolong morbidity and as the fever indicated a possible developing empyema, surgical treatment was advised.

On June 25 a week after onset, a left thoracotomy was performed. The left pleural cavity contained 1.2 liters of blood stained serum and 0.8 liters of blood clot. These were removed together with a film of fibrin over the lung, which then

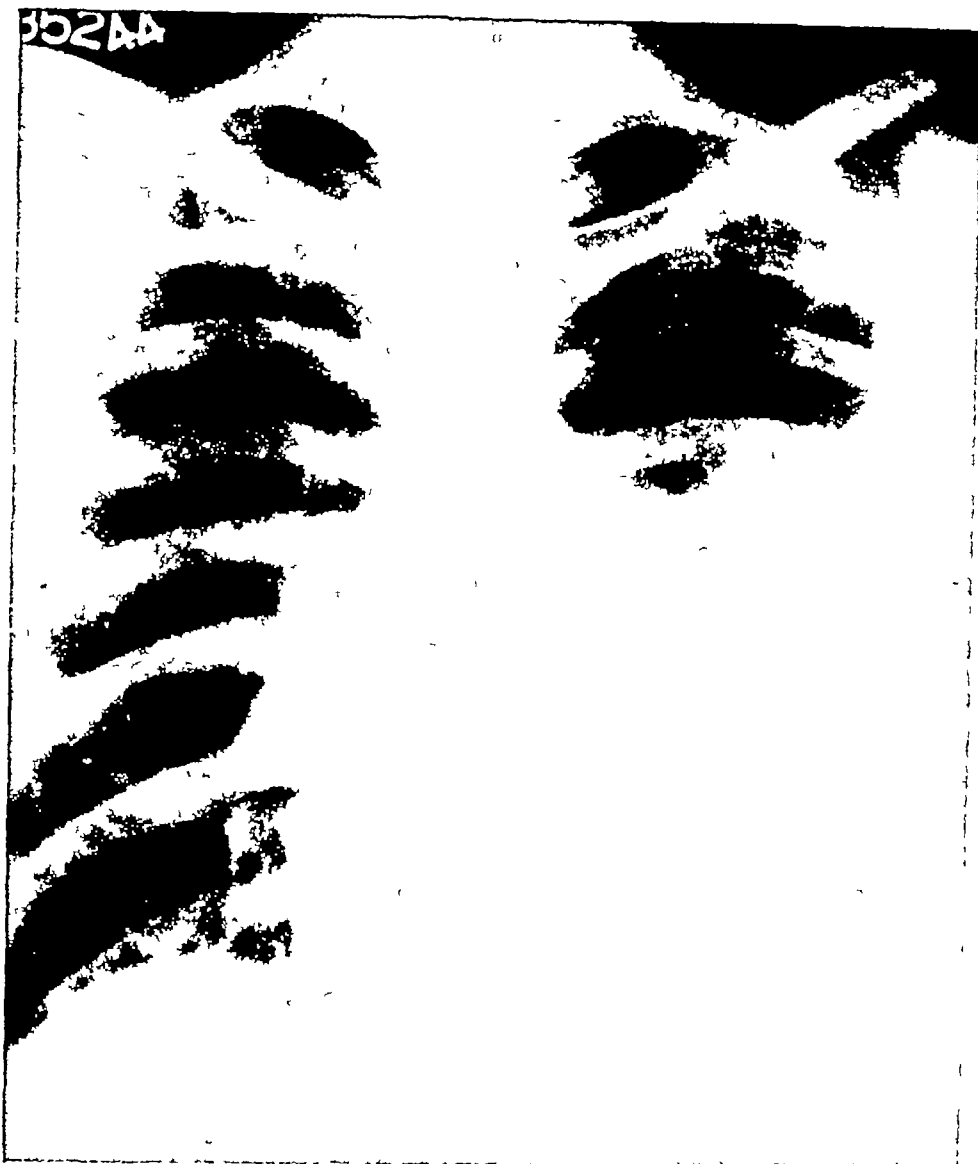


Fig 3B Two days later (June 25, 1954), no change, despite thoracentesis Thoracotomy, with decortication of left lung, was done

completely re-expanded At the apex of the lung were small bullous cysts beyond an old healed apical scar One cyst had torn and bled The cysts were excised, the lung sutured with continuous catgut, and the chest wall closed over water-seal drainage

The lung remained fully re-expanded, and the patient was discharged convalescent on July 5, i.e. 12 days after admission and 19 days after onset of his lesion

### CONCLUSIONS

In conclusion, spontaneous idiopathic hemopneumothorax is almost exclusively confined to males between the ages of 17 and 40 It usually occurs as a result of perforation of a bullous cyst that gives rise to bleeding from its wall, or from a torn pleural adhesion The most common symptoms are chest pain and increasing dyspnea and shock from blood loss The morbidity is high from fibrothorax, and the mortality is approximately 12 per cent. The treatment of choice is primary thoracotomy which not only is a life-preserving operation but also lowers morbidity. Late sequelae require decortication

## REFERENCES

- 1 Laennec, R T H. A Treatise on Mediate Auscultation and on Diseases of the Lungs and Heart, London, H Baillière 1846 460
- 2 Whittaker J T Case of hemotopneumothorax relieved by the aspirator Clin. Cincinnatl 10 193 1876
- 3 Pitt, G N A case of rapidly fatal hemopneumothorax, apparently due to rupture of an emphysematous bulla, Tr Clin Soc. London, 33 95 1900
- 4 Rolleston H D A case of fatal hemopneumothorax of unexplained origin, Tr Clin. Soc. London, 33 90 1900
- 5 Elrod, P D., and Murphy J D Spontaneous hemopneumothorax treated by decortication a case report, J Thoracic Surg. 17 401 1948
- 6 Seley G P., and Neuhoof, H Pulmonary decortication for clotted hemothorax following idiopathic spontaneous hemopneumothorax, J Thoracic Surg., 21 600 1951
- 7 Myers, R. T., Johnston, F R., and Bradshaw H H Spontaneous hemopneumothorax Report of a case treated by thoracotomy Ann. Surg., 133 413 1951
- 8 Ross, C. A. Spontaneous hemopneumothorax, J Thoracic Surg., 23 582, 1952.
- 9 Borrie, J Emergency thoracotomy for massive spontaneous hemopneumothorax, Brit. M J., 2 16, 1953
- 10 Fry W., Rogers, W L., Crenshaw G L. and Barton H. C. The surgical treatment of spontaneous idiopathic hemopneumothorax, Am. Rev. Tuberc., 71 30 1955
- 11 Calvert, R. J., and Smith, E. An analytical review of spontaneous hemopneumothorax, Thorax, 10 64 1955
- 12 Rusby N L Spontaneous pneumothorax, British Encyclopedia of Medical Practice, In term Suppl., 60 10 London, Butterworth, 1947
- 13 Hartzell, H. C. Spontaneous hemopneumothorax report of 3 cases and review of literature, Ann. Int. Med. 17 496 1942
- 14 Goldman, A. and Roth, H. Spontaneous pneumothorax A report of three unusual cases, Ann. Int. Med., 21 1011 1944
- 15 Helwig, F C. and Schmidt, E. C H Fatal spontaneous hemopneumothorax Review of the literature and report of a case, Ann. Int. Med., 26 608 1947
- 16 Freedman, P., Levine, S., and Solis-Cohen, L. Hemothorax In blood dyscrasias Am. J M Sc. 205 692, 1943
- 17 Dicara, L. V A rare case of spontaneous hemothorax, Am. Rev. Tuberc., 71 755 1955
- 18 Cosgriff S W Study of the coagulation mechanism of pleural blood in hemopneumothorax, Am. J Med., 8 57 1950
- 19 Clyne, A. J., and Hutter F H. D Spontaneous hemopneumothorax A surgical emergency Brit. M. J., 1 1058 1955
- 20 Read, C. T., and Berry F B The utilization of streptokinase streptodornase in a patient with hemopneumothorax and a patient with postpneumonectomy sanguinous coagulum, J Thoracic Surg. 20 384 1950
- 21 Shanda, W C. and Johnston, J H. Anaphylactic shock from intrapleural streptokinase-streptodornase, J Thoracic Surg., 31 320 1956



## TRAUMATIC CHYLOTHORAX

**Historical Note.** "Injury to the thoracic duct in the chest and the ensuing escape of chyle into the free pleural space constitutes a grave catastrophe in which there is scarcely an even chance of recovery" (1)

Ascellius in 1627 first described the lymphatic system (2) Six years later, Bartelot gave an account of chylous effusion into the pleural cavities (3) The first authenticated case of traumatic chylothorax was reported by Quincke (4) in 1875 In 1935 Lillie and Fox (5) stressed that careful analysis of the aspirated fluid is essential in making the diagnosis

Regarding treatment, intravenous chyle was introduced by Oeken in 1908 (6) and first described in the English literature by Williamson in 1920 (7) In 1931, van Nuys (8) reported using roentgen therapy, while in 1943 (9) Peet pioneered intrasternal injection of chyle In 1944, Loe (10) successfully treated a case caused by a gunshot wound through the upper chest and the root of the neck by thoracic duct ligation Lampson (1) in 1946 and Baldrige and Lewis (11) in 1947 each proved that the lesion could be cured by ligation of the thoracic duct within the chest; this is now accepted as the logical approach to treatment

**Anatomy of the Thoracic Duct.** The anatomy of the thoracic duct has been described by many different authors It shows numerous variations in structure, even in normal individuals Davis (12) describes 9 types, Anson (13) shows 12 These vary all the way from a single trunk to completely symmetrical bilateral structures Stuart (14) felt that a single channel might almost be regarded as an abnormality, so frequently is this part of the lymph system found to branch and subdivide

Whether single or branching, this duct, whose walls are composed of smooth muscle and fibrous tissue, varies from 4 to 6 mm in diameter It arises in an irregular, saclike dilatation, the cisterna chyli, on the anterior surface of the second lumbar vertebra and between the right crus of the diaphragm and the right side of the aorta There are tributary trunks—the intestinal, the bilateral lumbar, and the descending trunk—draining the lymph from the lower six intercostal spaces Entering the thorax through the aortic hiatus in the diaphragm, the thoracic duct is usually described as ascending on the vertebral column, behind the intercostal arteries, and between the azygos vein and the aorta At the fifth thoracic vertebra, it inclines to the left into the superior mediastinum, to ascend behind the aortic arch and the left subclavian artery, and between the left side of the esophagus and the left pleura.

In the neck, the thoracic duct, arching above the clavicle, crosses in front of the left subclavian artery, the vertebral vessels, and the thyrocervical trunk (or its branches), to enter the venous system at the junction between the left subclavian and the right subclavian vein at its junction with the left subclavian and the left internal jugular veins.

If there is also a *right* duct, it will be about 1.25 cm long and normally ends in the right subclavian vein at its junction with the left subclavian and the left internal jugular veins

Stranahan and associates (15) from an original study of thoracic ductography reported that in 30 cases studied the anatomic variations fell into five groups

- Group 1 An entirely right-sided duct—13 per cent,
- Group 2 Duct single throughout its course—30 per cent
- Group 3 Duct single at and below level of the tenth thoracic vertebra with a double duct or 'insular' pattern in the upper thoracic region—33 per cent
- Group 4 Many channels at the diaphragm but single trunks above—16 per cent
- Group 5 Two distinct ducts at and below the level of the tenth thoracic vertebra—16 per cent.

**Physiology** The thoracic duct conveys chyle, lymph, and lymphocytes into the systemic circulation. Its rate of flow varies from 60 to 90 ml per hour (16) so that if the duct were severed a patient could theoretically lose as much as 2 or 3 liters per day. The pressure within the duct is normally low, but after ligation it may rise to as much as 35 cm of water (11). The same factors which affect venous return influence the flow of chyle in the thoracic duct.

The chief characteristics of chyle are that it is milky in appearance, odorless and opalescent with a distinct creamy layer on standing which results from finely emulsified fat globules. It is sterile, resists putrefaction, is alkaline, has a specific gravity of 1.010 to 1.021, with a high fat content of 0.4 to 4 per cent, total protein exceeding 3 gm. per 100 ml. and total solids greater than 4 per cent. It is also rich in lymphocytes.

In chylothorax there is a progressive fall in lymphocyte percentage of the blood. With Sudan III its lipid substances are stained orange.

**Bacteriology** Many observers (1, 16, 17) support the view that chyle is bacteriostatic.

**Pharmacology** Vagal stimulation produces contraction of smooth muscle and a decrease in the flow of chyle through the duct. Adrenalin produces dilatation of the duct and an increased flow (16).

## PATHOLOGY

Traumatic chylothorax is an unusual disorder. Lampson (1) found only 69 cases recorded in the accessible literature. The formidable mortality, however, and the ease with which it can be effectively controlled by prompt treatment warrant description here.

In Lampson's series the age ranged from 9 to 62 years, but younger children are not immune. Sakula collected 13 cases in patients under 1 year of age and 5 within the first 4 weeks of life.

**Etiology** The causes fell into three large groups

1. *Closed trauma*—with or without fractured ribs, vertebrae, or clavicles. This group includes compression of the trunk, contusion of the back or chest wall or even a blow on the abdomen (20) as well as coughing, heavy lifting or hyperextension of the spine.
2. *Operative trauma*—such as sympathectomy (21), thoracotomy, excision of mediastinal tumors, operations on the esophagus or for the tetralogy of Fallot.
3. *Penetrating wounds*—stab or gunshot wounds.

The chyle accumulates in the posterior mediastinum, but sooner or later it erodes the overlying pleura and escapes into one or other of the pleural cavities

There is always a latent period between the time of injury and the onset of symptoms usually of three to seven days, though it may be months (22, 23)

As a general rule, injuries low in the thorax result in right chylothorax, and high injuries in left chylothorax

**Morbidity.** A tremendous volume of chyle can be lost, and thoracentesis may be required for many months Bauersfeld's (24) patient lost 22,275 ml of chyle by aspiration in 16 days Gordon's (23) case, admitted to hospital in August 1937, required monthly aspirations until December 1938, some 16 months later

**Mortality.** In analyzing 58 cases, Lampson found the mortality was 45 per cent, *primarily* due to respiratory or cardiac embarrassment or severe malnutrition "The patient bleeds to death, his death being due to loss of lymph and chyle, rather than to loss of actual blood" (11) Surgical ligation, however, has brought about a marked decrease in mortality

### CLINICAL FEATURES OF TRAUMATIC CHYLOTHORAX

**Symptoms and Signs.** These fall into four distinct and important stages (25)

- 1 Accident shock,
- 2 Recovery and latent period of pleural effusion,
- 3 Profound collapse,
- 4 Asthenia, and inanition

Typically, following an injury which severs the intrathoracic portion of the thoracic duct, there is a short *first stage* of shock, ending in apparent recovery In the *second stage*, lasting from 2 to 10 days, except for extreme thirst and hunger, the patient feels improved

In the *third stage*, with mounting quantity and pressure of intrathoracic chyle, there are signs of pleural effusion, collapse of the underlying lung, reduction of respiratory function, and perhaps a shift of the mediastinum As the quantity of chyle steadily increases, the patient becomes critically ill, with sudden onset of dyspnea, pale sweating facies, and even unconsciousness He may have glassy eyes and a weak, thready, racing pulse Following relief by thoracentesis, there is rapid recovery, but, depending upon the rate and quantity of chyle escaping into the pleural cavity, the pleural effusion re-accumulates and the symptoms recur.

In the *fourth stage*, through continued loss of chyle with its fluid, fat, and protein, there is progressive loss of weight and strength, with cyanosis and collapse

**Investigations.** There are three investigations

- 1 Roentgenograms of the chest to confirm the extent and effect of the effusion and the displacement of the mediastinum,
- 2 Thoracentesis A sample of the aspirate must be sent for laboratory identification, including estimation of fat content,
- 3 Klepser (26) advises that the patient eat a sandwich in which the dye has been mixed with butter The following day, on thoracentesis, the chyle, previously milky or blood-tinged, will be green, purple, or red, depending on the dye used, and will confirm that the duct is still leaking.

**Diagnosis** "Traumatic chylothorax should be considered as a possibility in every case of pleural collection of fluid due to surgical or accidental trauma to the neck, spine, chest or its contents" (18)

The rapidity of onset, the severity of symptoms and the equally rapid relief following thoracentesis are characteristic and distinguish this lesion from hemorrhagic or other types of shock. The pinkish gray "purulent" appearance of the fluid can readily be mistaken for empyema or hemothorax. Though rare complications such as chylopericardium and chylous ascites may occur in association with chylothorax.

### TREATMENT

There are three aims of treatment of this condition

- 1 To reduce the volume of chyle and so prevent pressure effects on the lung and cardiovascular system
- 2 To maintain nutrition,
- 3 To secure healing of the injured duct.

**Prevention of Mechanical Effects.** Chyle formation and flow is reduced by a low fluid intake and low fat diet. Its intrathoracic effect is reduced by repeated thoracentesis.

**Maintenance of Nutrition.** The patient should be weighed daily after thoracentesis. A strict fluid balance chart is kept. A high protein, high carbohydrate diet is taken and blood and plasma transfusions are given as required. Bed rest is essential to reduce the pumping action of respiration.

The idea of returning the chyle to the blood stream after aspiration from the pleural cavity at first sight, appears attractive. In 1942 Little and associates (22) reported returning 22 liters to the blood stream but this procedure has not been without mishap. For Whitcomb and Scoville (21) reported sudden death, anaphylactic in nature though Peet and Campbell (9) could establish no cause for a similar catastrophe. Skin sensitivity tests and the measuring of fat droplets (discarding chyle when the droplets have a diameter greater than three microns) should be used.

**Healing of the Injured Duct.** Regarding measures to promote healing of the torn duct Seaman (17) advises

- 1 Daily complete evacuant thoracentesis to avoid serious respiratory and cardiac embarrassment,
- 2 If, after seven days the leakage of chyle has not subsided, closed water seal catheter drainage with positive pressure suction should be instituted
- 3 If after an additional seven days this latter therapy does not produce definite progress exploratory thoracotomy is required.

### THORACOTOMY

The author feels that, because of the 50 per cent mortality that accompanies chylothorax, and the excellent results of early operation, it is useless to temporize. As soon as the diagnosis has been made and the patient made safe for thoracotomy this should be undertaken.

The affected pleural cavity is explored, the lung retracted, the mediastinal pleura inspected, and the fistula recognized. Further action depends on the findings.



lest empyema supervene. The normal apposition of visceral and mediastinal pleura may be all that is necessary.

- 4 If the lung does not fully re-expand nor the chyle fistula close thoracotomy and suture of the leak are essential.

## REFERENCES

- 1 Lampson, R. S. Traumatic chylothorax. A review of the literature, and report of a case treated by mediastinal ligation of the thoracic duct, *J Thoracic Surg.* 17 778 1948
- 2 Macnab D. S. and Scarlett, E. P. Traumatic chylothorax due to intrathoracic rupture of the thoracic duct, *Canad. M. A. J.*, 27 29 1932.
- 3 Olsen, A. M., and Wilson, G. T. Chylothorax, *J Thoracic Surg.* 13 53 1944 Quoting *Zeasas*, D. G. Die nicht operative entstandenen Verletzungen des Ductus thoracicus *Deutsche Zeitschr. f. Chir.*, 115 49 1912.
- 4 Quincke, H. Ueber fetthaltige Transsudate. Hydrops chylosus und Hydrops adiposus, *Deutsches Arch. klin. Med.*, 16 121 1875 Quoted by Macnab and Scarlett (ref. 2)
- 5 Lillie, O. R., and Fox, G. W. Traumatic intrathoracic rupture of the thoracic duct with chylothorax, *Ann. Surg.*, 101 1367 1935
- 6 Oeken. Ein Fall von Zerreissung des Ductus thoracicus infolge Brustquetschung, *München. med. Wchnschr.*, 55 1182, 1908
- 7 Williamson C. S. Case of chylothorax, with an analysis of pleural fluid by Professor U. H. Welker *Internat. Clinic*, 4 7 1920
- 8 Von Nuy, R. G. Chylothorax. Report of a case, *California & West. Med.*, 34 269 1931
- 9 Peet, M. M., and Campbell K. N. Massive chylothorax following splanchnicectomy. Treatment with intravenous and intrasternal transfusions of chyle, *Univ. Hosp. Bull., Ann Arbor* 9 2, 1943
- 10 Loe, R. H. Injuries to the thoracic duct. Report of a case of chylothorax in which the patient recovered after ligation of the thoracic duct, *Arch. Surg.*, 53 448 1946
- 11 Baldridge, R. R., and Lewis, R. V. Traumatic chylothorax. A review of the literature and report of a case treated by ligation of the thoracic duct and cisterna chyli, *Ann. Surg.*, 128 1056 1948
- 12 Davis H. K. A statistical study of the thoracic duct in man, *Am. J. Anat.*, 17 211 1915
- 13 Anson, B. J. *Atlas of Human Anatomy* Philadelphia, W. B. Saunders Co., 1950
- 14 Stuart, W. J. Operative injuries of the thoracic duct in the neck, *Edinburgh M. J.*, N.S., 22 301 1907
- 15 Stranahan, A., Alley R. D. Kausel, H. W., and Reeve, T. S. Operative thoracic ductography *J Thoracic Surg.*, 31 183 1954
- 16 Drinker C. K., and Vofsey J. M. *Lymphatics, Lymph, and Lymphoid Tissue*, Cambridge, Mass. Harvard University Press, 1941
- 17 Brit, A. B., and Connolly N. K. Traumatic chylothorax. A report of a case and a survey of the literature, *Brit. J. Surg.* 39 564 1952.
- 18 Seaman, J. B. Rationale and a new surgical technique in traumatic chylothorax, with report of a case, *J Thoracic Surg.*, 27 529 1954
- 19 Sakula, J. Chylothorax in the new born, *Arch. Dis. Childhood*, 23 240 1950
- 20 Baron, E., and Reardon, M. Chylothorax following a blow on the abdomen, *J Thoracic Surg.*, 28 11 1954
- 21 Whitcomb B. B. and Scoville, W. B. Postoperative chylothorax sudden death, following the infusion of aspirated chyle *Arch. Surg.*, 45 747 1942.
- 22 Little, J. N., Harrison, C., and Blalock, A. Chylothorax and chyloperitoneum. Effects of re-introduction of aspirated chyle, *Surgery* 11 392, 1942.
- 23 Gordon, J. Traumatic chylothorax, case report, *Ann. Int. Med.* 13 998 1940
- 24 Baversfeld, E. H. Traumatic chylothorax from ruptured thoracic duct, *J.A.M.A.* 109 16, 1937
- 25 Whiteside, W. C. Stewart, W. D., and Cuthbertson, A. N. Traumatic chylothorax, *Canad. M. A. J.* 61 374 1949
- 26 Klepner R. In discussion, Stranahan and others, reference 15
- 27 Meade, R. H., Hend, J. R., and Moen C. W. The management of chylothorax, *J Thoracic Surg.*, 19 709 1950.

## THE MANAGEMENT OF ACUTE EMPYEMA

**Introduction.** Although the incidence and severity of acute pleural empyema have fallen remarkably in the past two decades, nevertheless the appearance of antibiotic-resistant organisms makes it by no means certain that this state will continue. It is therefore felt justifiable to discuss in detail the management of acute empyema and its bearing on chronic empyema.

Empyema confronted and baffled physicians from the time of Hippocrates until the development of anesthesia, antisepsis, and roentgenography. The desire to drain the chest was offset by the ill-understood problems of pneumothorax resulting from such drainage.

The principle of "closed" intercostal drainage with water seal was introduced by Kenyon (1) in 1911. "Open" drainage had been practiced for centuries, but in 1918 Graham and Bell (2, 2a), faced with the prohibitive mortality from empyema accompanying the great epidemic of pneumonia of that year, showed experimentally that the mediastinum is so mobile that any increase of pressure in one pleural cavity from pneumothorax pushes it over into the opposite one, so that both lungs are equally compressed. They showed conclusively that, in treating these cases of synpneumonic streptococcal empyema, safe management required repeated aspiration until adhesion formation localized the empyema sufficiently for rib resection and drainage.

During World War II, further advances in management were made. With the introduction of antibiotics (3), positive pressure anesthesia to control mediastinal movement, the safe use of wide thoracotomy for decorticating infected hemothoraces (4), the application of the principles of water-seal drainage and the better appreciation of lung function, the dangers that Graham and Bell foresaw were overcome. Not only can the acute episode be largely controlled by careful aspiration and chemotherapy, but full lung re-expansion can be obtained by formal decortication, and any causative lung pathology, as bronchiectasis or neoplasm, may be removed by resection.

Modern treatment, therefore, aims not only to save the patient's life but also to

1. Eliminate infection,
2. Seek and treat its cause,
3. Produce full lung re-expansion,
4. Avoid a rigid chest wall with its secondary postural deformities,
5. Prevent chronic empyema.

### PATHOLOGY

Empyema thoracis is an abscess within one or both pleural cavities. It is not primarily a disease entity in itself but is always secondary to some other cause.

**Causes of Empyema.** The chief cause of empyema is lung sepsis, usually induced by lobar pneumonia. In spite of the introduction of chemotherapy and antibiotics, lung sepsis probably is still the most common cause. Empyema may develop from pleurisy during the acute attack of pneumonia (synpneumonic empyema), or following the pneumonia (metapneumonic empyema). It may also follow a bronchopneu-

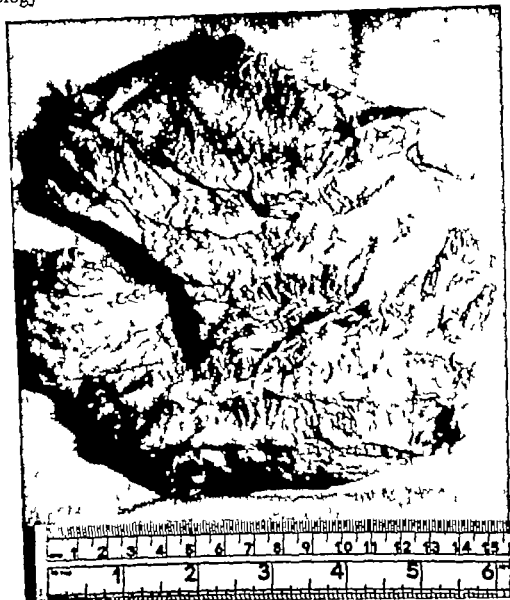


Fig. 1 Organized fibrin, 2.5 cm. thick, forming vice that cripples lung ventilation. Decortication specimen.

monia based on bronchiectasis lung abscess, infected lung cyst or blocking of a bronchus by a foreign body or by a neoplasm either benign or malignant.

Rarer causes include penetrating wounds of the chest wall postresection bronchopleural fistulas and perforation of the esophagus be it spontaneous (postoperative or the result of a penetrating foreign body) or from upward spread of a subphrenic or perinephric abscess.

No age group is immune. In the author's experience males are more commonly affected than females. Though usually unilateral empyema can be bilateral. It is associated with a variety of infecting organisms: pneumococci streptococci staphylococci rarely *B. coli* Friedländer's bacillus or anaerobic organisms. Chronic empyema due to the tubercle bacillus and to actinomyces is not included here.

Pleural infection is at first accompanied by the outpouring of a thin effusion filled with bacteria. This fluid rapidly becomes turbid, thickening and forming pus.

At first, with no pleural adhesions, the condition is essentially a diffuse suppurative pleurisy. The effusion however is rich in fibrin which is deposited in thick layers on the parietal and visceral pleura and diaphragm. The underlying lung tissue, held captive by the fibrin vice becomes compressed and nonfunctioning (Fig. 1).







Fig. 2C. Lateral film showing bilocular empyema.

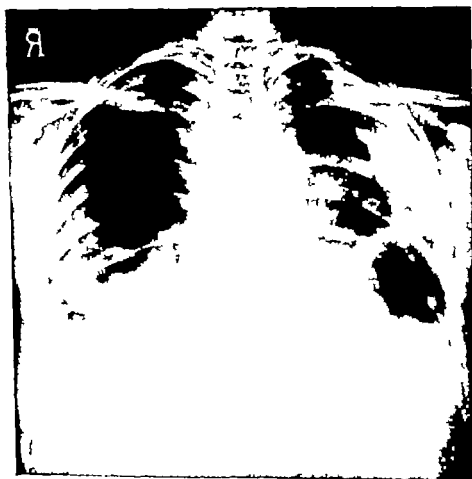


Fig. 2D. Bilateral empyema.

The empyema may be *total*, filling the pleural cavity completely, and may even have tension phenomena with shift of the mediastinum. When patients are nursed in the sitting position, however, empyema is usually *basal* and posterior. Following aspiration, it may loculate in any part of the pleural cavity, causing such other varieties as a lateral, apical, interlobar, or bilocular empyema (Figs. 2A-D).

**Complications of Acute Empyema.** Complications are four in number

- 1 Bronchopleural fistula,
- 2 Empyema necessitans,
- 3 General toxemia producing
  - (a) Progressive anemia,
  - (b) Heart failure,
  - (c) Cerebral abscess;
- 4 Chronic empyema



Fig 3A. Roentgenogram with typical fluid level of bronchopleural fistula

*Bronchopleural Fistula* (Fig 3A) This arises in one of two ways

1. A neglected or unsuspected empyema masked by antibiotics penetrates the lung parenchyma and ruptures into a bronchus or
2. A lung abscess, secondary to a blocked bronchus bronchiectasis or suppurative pneumonitis ruptures into the pleural cavity

The result is a "putrid empyema" with virulent mixed pyogenic organisms including *B. coli* producing brownish foul smelling pus. The dangers are total empyema, tension pyopneumothorax and drowning by aspiration of pus through the fistula into the lungs.

*Empyema Necessitans* This is the name given to an empyema which presents subcutaneously.

In neglected cases the empyema may track backward between the ribs and then down in the paravertebral tissue planes to present as a subcutaneous lumbar abscess or it may pass forward along with the intercostal vessels and appear on the ventral thoracic or abdominal wall.

*General Toxemia* Pleural sepsis rapidly causes weight loss and a severe progressive microcytic hypochromic anemia. Especially in the elderly signs of heart



Fig. 3B. Bronchogram of patient with left chronic empyema, destroyed left lung and bronchopleural fistula.

failure with ankle edema may follow, and even auricular fibrillation. Metastatic abscesses, especially cerebral abscess, may also occur occasionally in neglected cases.

**Chronic Empyema** (Fig 3B) This is one which, after six to eight weeks of treatment, is not steadily healing. It is the most common complication of acute empyema and as such is preventable. Recognition and treatment must be prompt, for a "week's delay in the early stages means a month's longer convalescence" (5).

The most common causes are.

- 1 Delay in recognition of acute empyema,
- 2 Delay in starting adequate treatment,
- 3 Faulty equipment and inadequate aspiration,
- 4 Too much reliance on antibiotics which remove the toxemia but leave the space,
- 5 Delay in consulting with a surgeon
- 6 Inadequate initial drainage

The patient's best chance of cure depends on surgical consultation as soon as the diagnosis is made and complete continuity in treatment from then until the space is finally obliterated.

## CLINICAL FEATURES

**Types of Onset and Physical Findings.** Empyema, once a readily recognizable disorder, in these antibiotic times is fast becoming one of the great imitators. Further, when assessing the patient we must constantly ask and answer the question, "What is this empyema due to?"

When an acute empyema follows a pneumonia not treated by antibiotics or is caused by organisms resistant to antibiotics, signs of intrapleural fluid are added to those of the pneumonia. This may occur during or following the pneumonic episode. If the latter, then the temperature does not fall to normal as the pneumonia resolves, but remains elevated.

When, however, antibiotic drugs have been given, the clinical course of the pneumonia and of the empyema is usually so altered that the empyema is often detected only late in the illness. Many patients do not show the classical picture, and many have few signs and symptoms. The effusion may even go undetected until an established bronchopleural fistula demands urgent relief. Then there are signs of emaciation and anemia, together with copious, foul-smelling sputum draining from the mixed infection in the empyema space. Physical signs again show fluid. The rapid decline in health can be misdiagnosed as pulmonary tuberculosis or bronchial carcinoma, only after full investigation, including thoracentesis, is the true diagnosis established.

In children, especially with staphylococcal pneumonia, the sudden onset of a tension pyopneumothorax from rupturing of a superficial abscess into the pleural cavity creates a profound emergency. The signs of pneumonia are suddenly altered to those of tension pneumothorax, extreme breathlessness, even cyanosis, hyper-resonance on the affected side, and compression of the sound lung by intrathoracic tension displacing heart and mediastinum (Figs 4A-C).



Fig. 4A Infant with staphylococcal pneumonia in right lung.

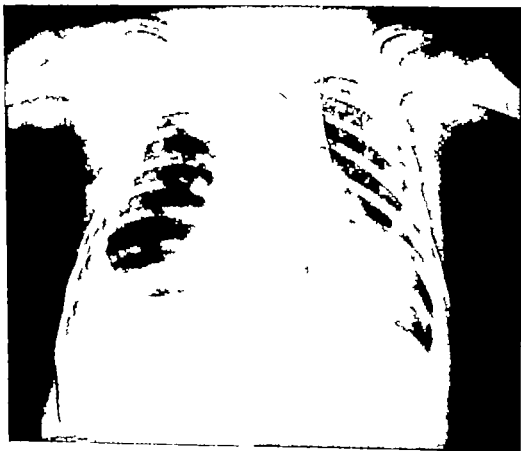


Fig. 4B Same patient with tension pyopneumothorax from intrapleural rupture of small sub-pleural abscess.



Fig 4C Same patient, cure by intercostal water-seal drain

When the empyema follows spontaneous rupture of the esophagus or the rare leaking anastomosis after esophageal operations, the patient also has signs of fluid and air in the pleural cavity (Figs 5A and B)

**Investigations.** There are four important steps in diagnosing and treating acute empyema

- 1 Careful history taking and physical examination,
- 2 Chest roentgenograms,
- 3 Thoracentesis and aspiration of pus,
- 4 Finding the cause

*History taking and physical examination* have already been discussed under Clinical Features

**Roentgenography in Acute Empyema** An uncomplicated acute empyema has a characteristic roentgenogram of fluid in the pleural cavity with a shadow obliterating the costophrenic sinus and arching outward and upward in a curve toward the axilla. Lateral films will localize the fluid

When the empyema has become encapsulated, roentgenograms may simulate a lung abscess or tumor. The *tension pyopneumothorax* of children and its effects are obvious. With a *bronchopleural fistula* there is a distinct fluid level. Check should, however, always be made to determine if the patient has not already had a *thoracentesis without a two-way tap*, thus letting air into the chest from outside



Fig 5A. Roentgenogram of patient referred with right total empyema from leaking esophageal anastomosis.

*Thoracentesis* The value of thoracentesis is fourfold

- 1 It confirms the presence of fluid
- 2 It determines whether or not the fluid is purulent
- 3 It provides material for culture
- 4 It is usually the first step in treatment.

Thoracentesis follows careful clinical and x ray localizing of the lesion. *Fluoroscopy* is helpful in deciding the exact site at which to aspirate. The technic is described in Chapter 2.





Fig 5B Lung re-expansion after intercostal water-seal drainage

A 10 to 20 ml specimen of pus is sent for aerobic and anaerobic culture of the organisms, and for determining their sensitivity to antibiotics. If antibiotics have already been given, however, the pus may be sterile.

After the specimen is collected, formal aspiration proceeds (see below) until the space is empty. Thereafter, 1 million units of penicillin in 10 ml of saline are left in the space.

The operator must not allow any air to enter from outside, as this will precipitate collapse of the lung, separation of the pleural layers, tension pyopneumothorax, and acute respiratory distress (Fig 2B). Such an emergency can be adequately relieved only by prompt intercostal water-seal drainage.

## MANAGEMENT OF ACUTE EMPYEMA

The introduction of antibiotic agents over the past 20 years has revolutionized the management of acute empyema. Not only is the incidence of empyema following pneumonia greatly reduced, but it is occasionally possible to effect a cure of the empyema by aspiration alone. Each such case, however, must satisfy all the aims of treatment already mentioned, for the price of failure is chronic empyema.

Briefly the treatment of an acute empyema varies with the stage of the disease. The early phase of diffuse suppurative pleurisy requires antibiotic therapy with aspiration. The later phase of a localized empyema requires surgical drainage. This may be enough but when the lung does not fully re-expand or is found to contain correctable disease then pulmonary decortication with or without resection is finally required.

- ① **Antibiotic Therapy** Culture of the diagnostic aspirate establishes the nature of the infection, and the sensitivities of the organisms to antibiotics. The appropriate one is given systemically and injected locally after each therapeutic pleural aspiration. Once the space is sterilized or drained, however, this is discontinued.
- ② **Aspiration. PRINCIPLES** Combined with antibiotics, aspiration aims to (a) sterilize the space and (b) keep it obliterated so that the parietal and visceral pleura will adhere firmly together.

For this procedure to succeed, the empyema must be aspirated as completely as possible each day and a dose of the effective antibiotic must be left in the space before the needle is removed. At the outset, it must be fully appreciated that success depends on aspiration that provides the equivalent of free drainage.

**TECHNIC.** In adults a 50 ml. syringe with a wide bore needle is ideal whereas for children a 20 ml. syringe is advised. The needle, tap and syringe should have the Luer locking device and all should be thoroughly tested before use. The technical details are described in Chapter 2. As the aspiration may take from 20 to 30 minutes both patient and technician should be comfortably placed.

**FIBRINOLYTIC ENZYMES.** With this method, intrapleural fibrin clots may be liquefied by fibrinolytic enzymes (streptokinase and streptodornase), but, unless these enzymes are used early in the treatment, they will be useless. The dose is 10 000 units S.K. and 4 000 units S.D. injected intrapleurally after complete aspiration. Within a few hours there is a febrile reaction, the patient feels an uncomfortable tightness in the chest and needs prompt relief by aspiration the next day.

**LIMITATIONS OF ASPIRATION** Aspiration has several limitations. It is unlikely to cure a fully established empyema when it is multilocular or when the organisms are antibiotic resistant. Further while it may sterilize it may not obliterate but may leave a sterile empyema which produces all the physical deformities of a chronic fibrothorax. It is absolutely contraindicated when the empyema is due to esophageal perforation or is complicated by bronchopleural fistula or tension pyopneumothorax. For these more effective methods are urgently required.

Johnson and Kirby (6) rightly warn that "recognition of these limitations [of aspiration] is important. Persistence in utilizing this method of treatment when prompt improvement is not evident only leads to the development of chronic empyema. If the patient's temperature is not normal and the cavity is not well on the way towards obliteration after 7 to 10 days, surgical drainage should be instituted."

To summarize therefore aspiration therapy can succeed only in empyemas found early (i.e. within a week of onset), with thin pus, no thick fibrin rind on the

lung or in the pleural cavity, and when aspiration is diligent, daily, and complete and is checked by postaspiration fluoroscopy or roentgenograms

**Surgical Treatment.** In acute empyema, surgical treatment means drainage, either (a) intercostal drainage or (b) rib-resection drainage

**INTERCOSTAL DRAINAGE** The technic is described in Chapter 2. This is the simplest of thoracic surgical operations. It provides airtight drainage of the pleural cavity and is a true emergency method. In the management of acute empyema, intercostal drainage is indicated for immediate relief from the dangers of

1. Large pyothorax, especially with tension phenomena in children,
2. Empyema with bronchopleural fistula,
3. In desperately ill patients with a large neglected empyema,
4. Empyema secondary to leaking esophageal anastomosis,
5. As preliminary control of the empyema accompanying intrathoracic perforation of the esophagus

The advantages are that the method is quick, simple, effective, often lifesaving, inflicts minimal surgical trauma, and produces no open pneumothorax

Like aspiration, however, there are disadvantages. Except with small children, or with an early empyema containing thin pus, it is rarely completely effective alone. Fibrin clots cannot escape, in four to five days the tube frequently blocks, and at the time of the emergency the tube may not have been placed in the most dependent part of the pleural cavity. Within a week, therefore, intercostal drainage should be reassessed by roentgenograms and, if unsatisfactory, corrected by rib-resection drainage in a more favorable site (Figs 6A-C)



Fig 6A Acute empyema with bronchopleural fistula treated by intercostal drainage



Fig. 6B Symptoms relieved, but pleurogram reveals unsatisfactory drainage.



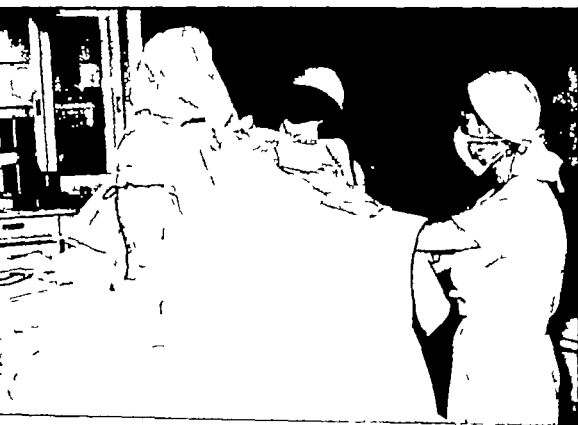
Fig 6C. Satisfactory dependent progress following rib-resection drainage.

**RIB-RESECTION DRAINAGE. *Time to Operate*** In uncomplicated empyema, operation is usually delayed until repeated chest aspiration and adhesion formation between the parietal and visceral pleurae have localized the empyema. In pneumococcal empyema, with its heavy outpouring of fibrin, this usually occurs within 10 days, whereas with streptococcal empyema it may take longer. As Romanis (7) wrote in 1924, "In general, whatever the organism, the longer the pus has been present in the pleura, the thicker it will be, and the greater will be the liability for the formation of pleural adhesions, thickened pleura and solid fibrinous masses." To delay, therefore, is to invite chronic empyema.

The site for correct dependent drainage is determined by studying posteroanterior and lateral chest roentgenograms. This can be aided by injecting radiopaque oil (5 to 10 ml) into the empyema space to define its lowest limit.

***Position.*** Usually, the patient sits on the operating table with his feet on a stool and his arms and head resting on a pillow placed on a Mayo instrument table (Fig 7). If too ill to sit up, he lies with his chest over a pillow on an inclined operating table, thus making the involved side more prominent and accessible. When the empyema is anterior, the patient lies on his back.

***Anesthesia*** Because of the dangers of bronchopleural fistula and endobronchial aspiration of pus, local anesthesia is advised, and it is injected into the subcostal groove of the selected rib, as well as into the two rib spaces above and the two below. Injection of 5 ml of 2 per cent lignocaine per intercostal nerve gives a safe, effective, rapidly acting anesthesia. The skin over the site of operation is next infiltrated with 0.5 per cent lignocaine with adrenalin solution (see Chapter 4, Fig. 5).



Figs. 7A and B. Position of patient for draining posterior empyema under local anesthesia.

*The Operation* (Fig 8) The empyema should be opened as low as possible by resecting portions of the ninth or tenth ribs in the posterior axillary line. A 6 cm vertical skin incision is made. This is certain to be over the empyema and can be enlarged downward if required. The underlying muscles are divided, and the periosteum is exposed and incised along the rib for at least 6 cm. The rib is next bared with a periosteal elevator by pushing the divided periosteum to the rib edges. The upper edge is cleared in one stroke by sweeping the periosteal elevator along it from behind forward. The lower edge requires the reverse action, and the posterior surface is cleared with the same elevator or a Doyen rib raspator. A 5 cm length of rib is resected, the bone ends protected with Horsley's wax, and the site of the empyema again verified by aspirating pus. The thickened parietal pleura is next incised. Pus now drains freely and is aided by coughing or suction. Fibrin clots are removed with a pair of sponge-holding forceps. A pleural biopsy is taken for histologic examination, and, if required, pus is aspirated for aerobic and anaerobic culture. Finally, the cavity is inspected with a malleable light and its lining walls wiped with gauze swabs on sponge forceps. A wide-bore rubber tube with several side holes is inserted, and the skin is approximated around it with nylon sutures which also anchor the tube.

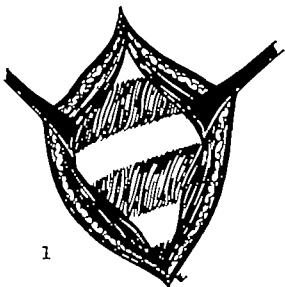
**BILATERAL EMPYEMA** This is not common, but the principles of its treatment are the same as for unilateral empyema except that one side only—the larger or the complicated empyema—must be drained first. The opposite side is controlled by aspiration as long as possible. Aspiration may suffice, as shown in Figure 9A; or rib-resection drainage (Fig 8) or decortication may later be required (Figs 9B and C).

**After-Care.** If the empyema space is large and there is a tendency to paradoxical movement, the tube is connected in the operating room to a water seal for closed drainage, and continuous suction to assist lung re-expansion is applied in the ward.

The patient is seated upright soon after his return to the ward, and an x-ray check is made of the chest and of the tube. If the latter is not satisfactory, it is adjusted. Subsequent management of the tube is the surgeon's responsibility, he must supervise all shortening until healing is complete.

When there is considerable purulent discharge, closed drainage is continued up to a fortnight or more. From a nursing standpoint, closed drainage is easier than open drainage. When the discharge lessens, however, the bottle is removed, and a short tube with side holes inserted, fixed to the chest wall with a safety pin and strips of adhesive plaster, and covered with cotton and an elastoplast corset (Chapter 2, Fig 5). As Allison (8) wisely points out, "any system of closed drainage tends to focus attention on the apparatus rather than the empyema cavity."

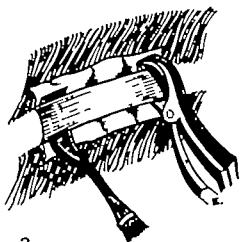
If there is no fistula, daily irrigation with eusol solution further aids healing, and, if pyocyanus infection is present, irrigation with phenoxytol 2 per cent is of value. Mason's (9) method of irrigating by keeping two tubes of different sizes in the wound and connecting the smaller to a funnel ensures that fluid readily escapes without building up excessive intrapleural pressure. Syringing with positive pressure should be avoided.



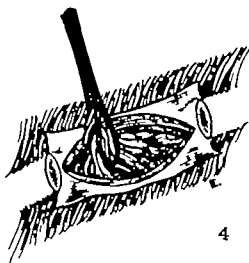
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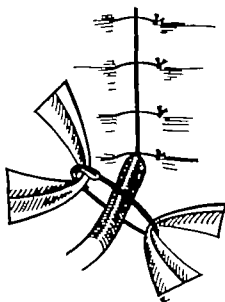
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Fig. 8 Steps in rib-resection drainage for empyema. 1 Ribs exposed by 6-cm. vertical incision. 2, Removing periosteum of the selected rib 3 Rib shears dividing the rib. 4 Sponge forceps removing fibrin clots. 5 Suction removal of residual pus. 6, Firm closure of skin edges around the drainage tube before connecting it to a water-seal drainage.



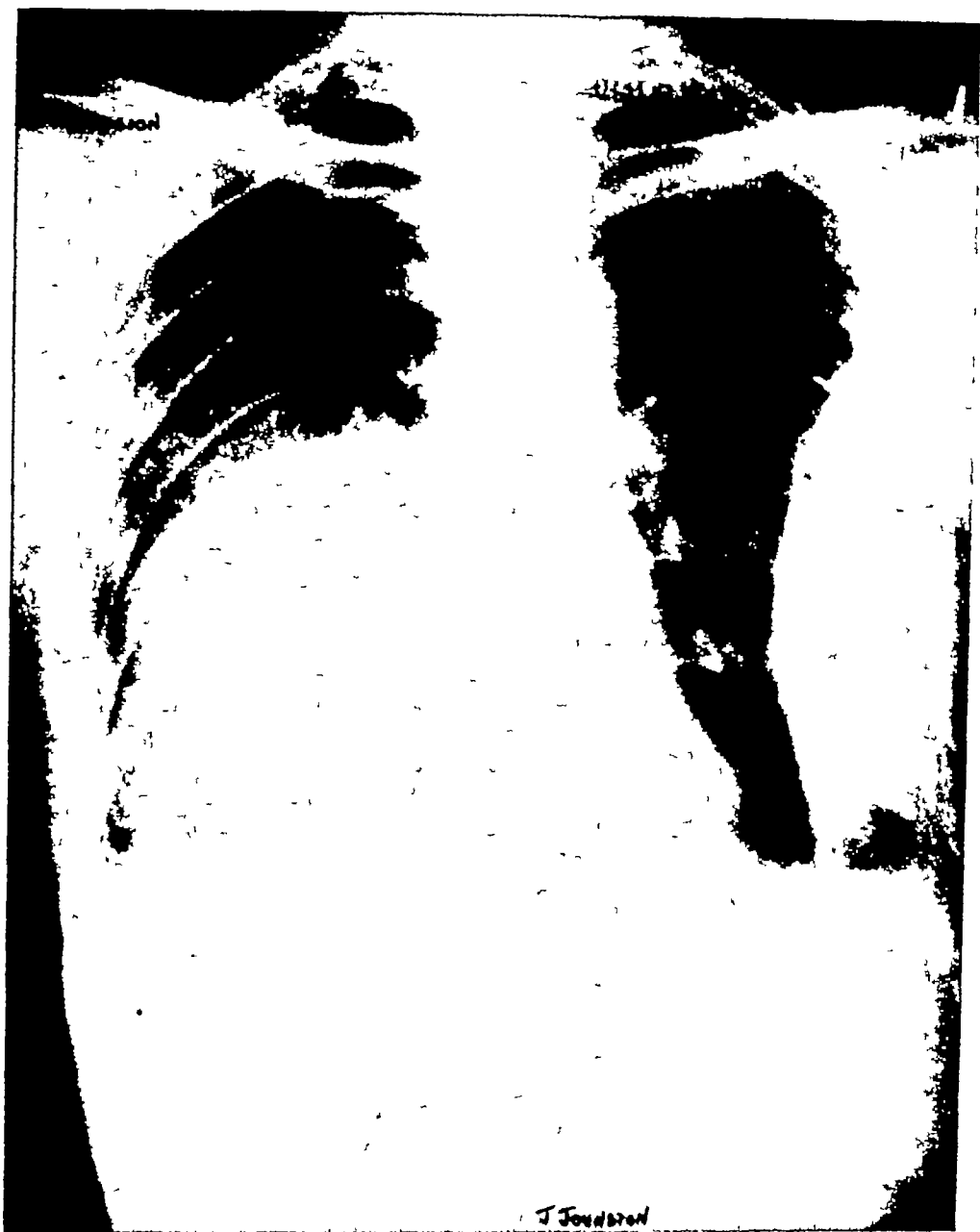


Fig 9A Bilateral empyema treated by aspiration on right side and decortication on left



Fig. 9B The result two weeks later

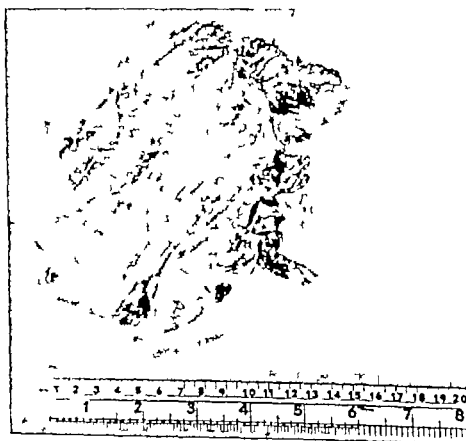


Fig. 9C. The decortication specimen.

**Progress** (Fig 10) At fortnightly intervals, the size of the empyema space is checked by pleurograms, during which radiopaque oil is instilled into the space, the opening plugged with cotton and marked, and films taken in two planes. As the space heals, it closes in around the tube which should be kept 2 cm short of the total length of the sinus. Each time the tube is changed, the length of the sinus is measured with a catheter or empyema sound and the tube adjusted. Only when the sinus has healed to rib level, as outlined by pleurograms and sounding, is the tube finally removed.

If at any time the tube slips out, it must be immediately sterilized and reinserted, for the sinus contracts in a matter of minutes and will require Hegar dilators to re-establish it.

**General Measures.** The following general measures assist healing.

- ✓1 The patient is encouraged to get up as soon as possible. Even when on closed drainage, he can carry his bottle about and take part in physical training classes.
- ✓2 *Deep breathing exercises* are important to encourage lung re-expansion and closure of the empyema space and must be done repeatedly throughout the day to be effective.
- ✓3 *Diet* Because of continued protein loss in the pus, high protein diet is required.
- ✓4 *Blood* Fortnightly blood examination is advisable. Iron in the form of coll-iron 4 ml thrice daily is given to correct the iron deficiency anemia. A hemoglobin level below 10 gm requires blood transfusion.

Acute empyemas treated by intercostal or rib-resection drainage are rarely healed in less than six weeks and may take from two to three months, especially in the elderly.

**Difficulties of Tube Management.** When a tube blocks, investigation will probably reveal one of the following causes (8)

- 1 *Obstruction by Fibrin Masses* Fibrin may have been left loose or on the walls of the cavity during drainage or may have formed after drainage. The fibrin acts as a nidus for chronic infection. In management, the tube is removed daily, sterilized, and put back, and the cavity at first is left strictly alone. When there is no bronchopleural fistula, eusol irrigations help. If the fibrin persists, exploration under anesthesia and removal of the fibrin with sponge-holding forceps is required.
2. *Obstructing Granulating Tissue* This grows into and blocks the tube, especially when the cavity has narrowed to a track and where regular tube toilet is not strictly observed.
- 3 *Obstruction by Kinking of Tube* This may occur in a tortuous sinus track or with closed water-seal drainage. It is avoided by using a tube of reasonable strength and also by seeing that the patient does not rest on any part of the tube leading to a drainage bottle.
- 4 *Tube Too Short to Reach Pleura* This occurs
  - (a) When an uninstructed nurse shortens the tube with every dressing,
  - (b) When the sinus has narrowed after a tube has fallen out and not been promptly re-inserted,
  - (c) When, with a self-retaining tube tethered to a bed, the patient suddenly turns and pulls the tube from the empyema cavity into the chest wall.

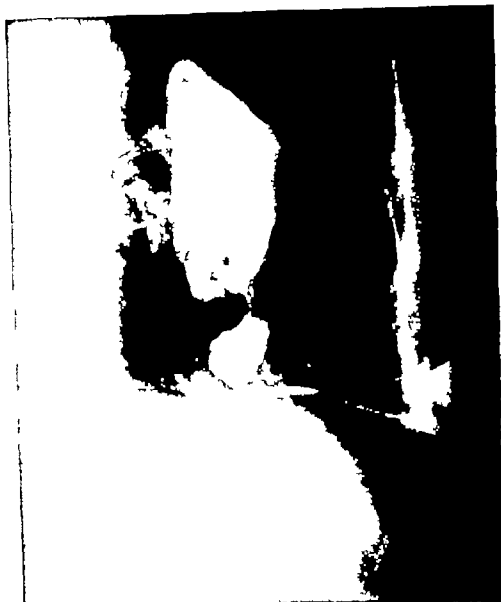


Fig. 10A. Poorly drained space requiring longer tube.

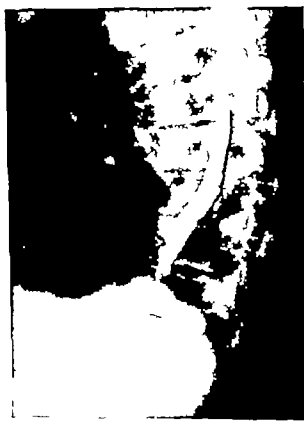


Fig. 10B. Five weeks later the space narrowed to tube track tube therefore could be shortened until closure of space completed.

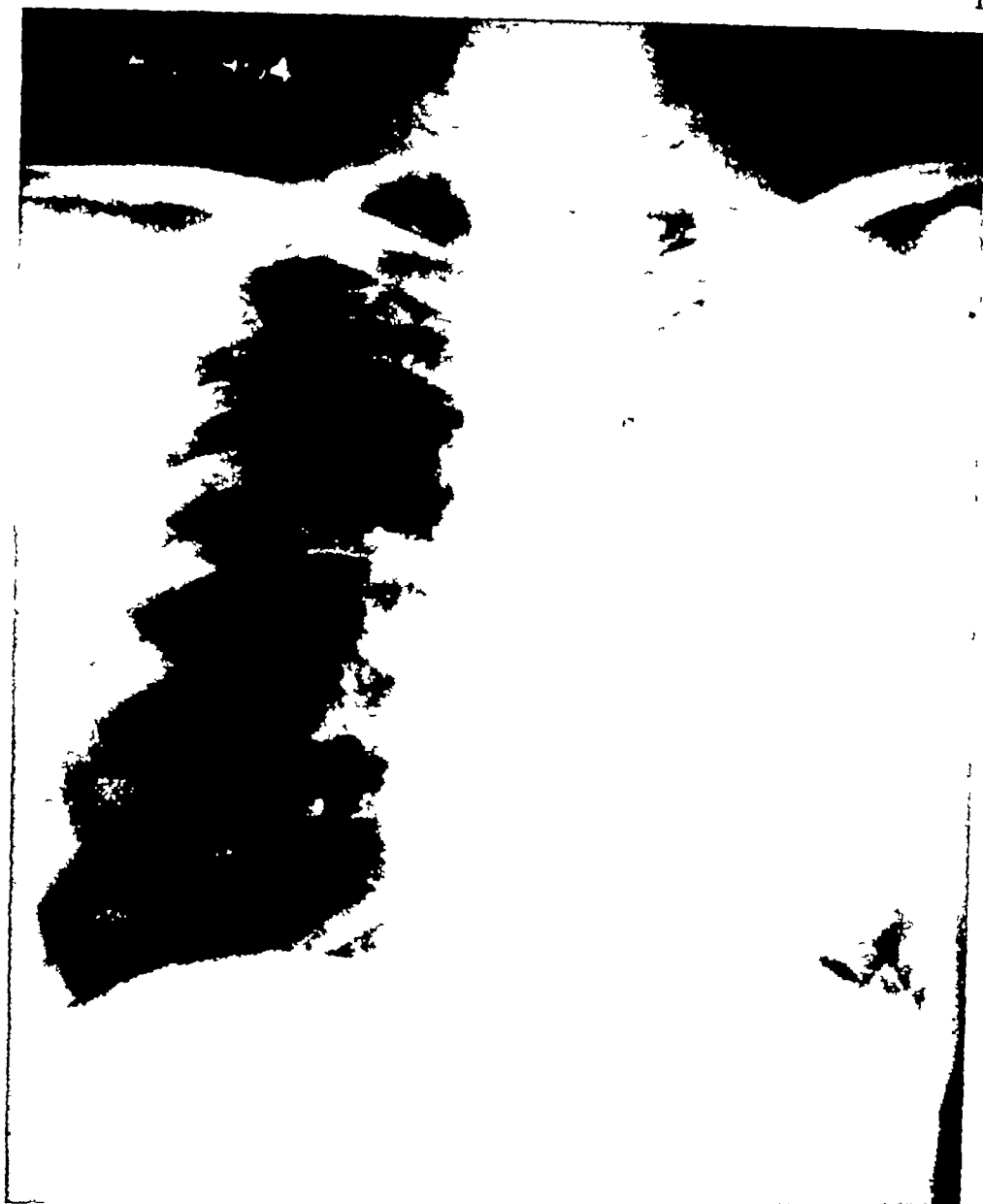


Fig 11A Bilocular left empyema treated by decortication For corresponding lateral film see Fig 2C.

- 5 *Tube Through the Chest Wall but Not Reaching the Cavity* This can happen during the healing of an apical empyema, when, as a result of elevation of the diaphragm and expansion of the lung, the base of the empyema may heal more rapidly until a cavity is left above, communicating with the tube by a long narrow track. Either the tube must be lengthened to enter the space, or else a second drainage opening established higher up.
- 6 *Tube Too Far Into Cavity* The end may be so far into the cavity that pus collects below it and only drains as an overflow. With open drainage, this is avoided by having a lateral hole where the tube enters the space. With closed drainage, a safety pin marker, placed at skin level after the tube has been pulled down, will ensure that it does not ride up again.
- 7 *Part of the Cavity Below the Drainage Opening.* When there is a broad-based empyema cavity and the anterior and posterior ends are lower than the middle, two drains—one in front and one behind—are required. With one drain only, there is always a puddle of pus that will never clear.

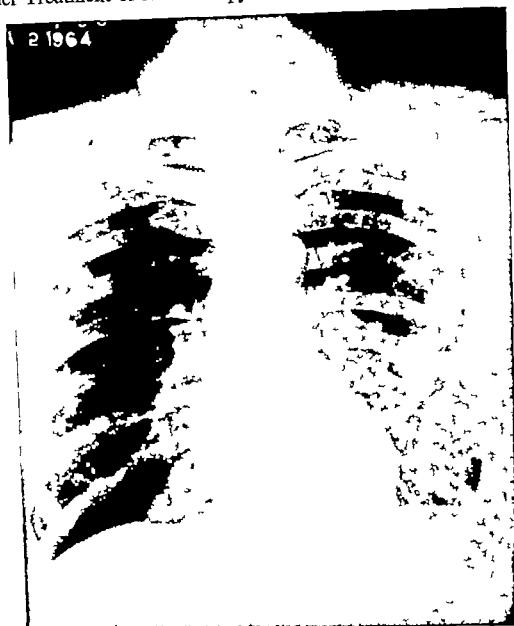


Fig 11B Same patient as discharged fig 17 days after operation.

- 8 *Loculation.* Rarely pleural empyemas become loculated, especially during the initial phase of aspiration. These loculi may be separate joined by a narrow sinus or have a wide opening between. They usually require separate drainage.

#### FURTHER TREATMENT OF ACUTE EMPYEMA

Antibiotic agents, by altering the course of acute empyema, have made possible two further methods of treatment. These are *decortication* and *resection*.

**Decortication.** In patients in whom aspiration has controlled the acute phase and intrapleural antibiotic agents have made the effusion sterile lung movement may still be grossly limited by fibrin lining both parietal and visceral layers of the pleura. Each layer may be as much as 2 cm thick. Unless removed, this fibrin will hold the lung firmly and permanently collapsed (Chapter 9 Fig 1). As modern aims of treatment include full lung expansion and the avoidance of a rigid chest wall, the fibrin vice must be removed. Provided, therefore, investigations are satisfactory and bronchoscopy and bronchography have revealed no permanent lung disease then decortication is the treatment of choice.



Fig 12A Left acute empyema relieved by intercostal drainage

The operative details are the same as those described under hemopneumothorax (p 112), and the secret of postoperative success lies in holding the decorticated lung fully expanded, with the pleural layers in contact by strong intrapleural suction

The operation is especially effective in young and middle-aged patients, and it should not be delayed beyond the time the empyema has been sterilized. A feature of successful decortication is the great reduction in length of postoperative care—to as little as a fortnight (Figs 11A and B)

**Pulmonary Resection.** In like manner, when investigation by bronchiography reveals lobar bronchiectasis or bronchoscopy reveals a neoplasm, then pulmonary resection is combined with decortication.



Fig. 12B. Subsequent bronchograms revealed partially re-expanded upper lobe and lower lobe bronchiectasis. The patient recovered after pleurolobectomy (Courtesy George A. Mason)

If for example, the disease is lower-lobe bronchiectasis or peripheral neoplasm decortication of the upper lobe combined with resection of the diseased lower lobe by the operation of pleurolobectomy is the treatment of choice (Figs. 12A and B). However, when the disorder is a total bronchiectasis or a central neoplasm, especially carcinoma, then treatment is pleuropneumonectomy (Figs. 13A and B).

Preoperative assessment requires complete aspiration and sterilization of the empyema cavity with antibiotics as well as the usual blood check, spirometry and so on. The operation commences with parietal pleural decortication, and thereafter consists of the standard resection required for the disease.

In the postoperative period, the appropriate antibiotics are given systemically and locally to ensure that no further empyema develops. After pneumonectomy this is most important and requires daily aspiration and instillation of antibiotics for at least 10 days after operation.





Fig 13A Acute empyema referred for drainage, bronchoscopy revealed carcinoma of lung causing collapsed lower lobe, recovery after pleuropneumectomy

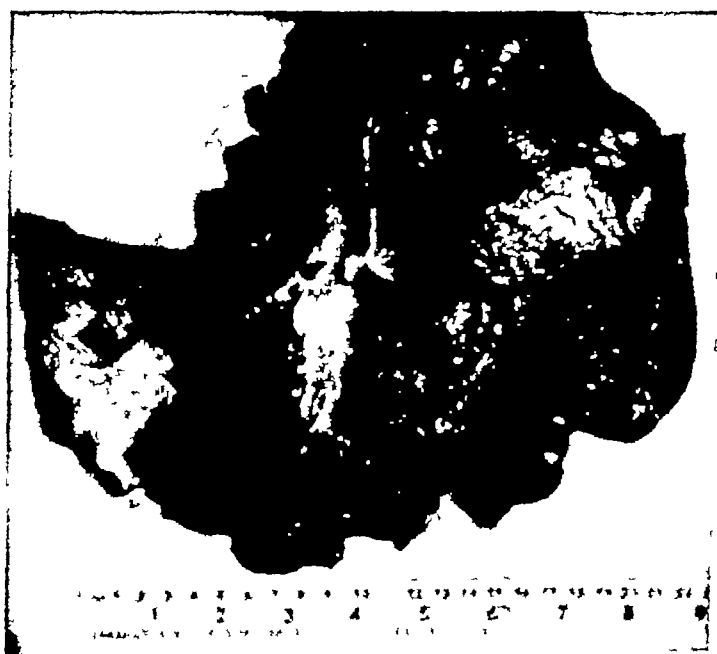


Fig 13B Specimen removed

## CONCLUSION

In conclusion, when the aims of treatment are fully realized at the outset and the "dangerous corners" appreciated, patients with an acute empyema, by having prompt, well-coordinated treatment, can be cured in minimal time and without the tedium and dangers of complications or the endless disappointments of a chronic empyema

## REFERENCES

- 1 Kenyon, J. H. A preliminary report of a method of treatment of empyema in young children, *M. Rec.* 80 816, 1911
2. Graham E. A. *Some Fundamental Considerations in the Treatment of Empyema Thoracis*, London, Henry Kimpton, 1925
- 2a ——— and Bell, R. D. Open pneumothorax. Its relation to treatment of empyema, *Am. J. M. Sc.* 156 839 1918
- 3 D'Abreu, A. L., Litchfield, J. W. and Thomson, S. Penicillin in the treatment of war wounds of the chest, *Brit. J. Surg.* 32 179 1944
- 4 Price Thomas, C., and Cleland, W. P. Decortication in clotted and infected hemothoraces, *Lancet*, 1 327 1945
- 5 Sellors T. H. Empyema, *Brit. M. J.*, 1 704 1952.
- 6 Johnson, J., and Kirby C. K. *Surgery of the Chest*, Chicago The Year Book Publishers, Inc. 1952.
- 7 Romanis W. H. C. On the treatment of empyema and acute abscess of lung, *Practitioner* 113 331 1924
- 8 Allison, P. R. Management of acute pleural empyema, *Brit. M. J.*, 2 383 1943
- 9 Mason, G. A. The post-operative management of acute empyema thoracis, *Brit. M. J.*, 2 1197 1934

# Pulmonary Emergencies

## 10

### THE MANAGEMENT OF ACUTE LUNG ABSCESS

**Introduction.** The same factors that have reduced the incidence of acute pneumonia and empyema have lessened the severity of most lung abscesses, so that now, when uncomplicated, such abscesses seldom present themselves as acute surgical emergencies. Nevertheless, because of the serious complications and emergencies that can, and do, arise if these conditions are neglected, and because so many lung abscesses ultimately require surgical treatment, their "dangerous corners" should be well appreciated and further treatment carried out in cooperation with a thoracic surgeon as soon as the diagnosis is established.

Historically, lung abscess was always a highly fatal disease. In 1924, Romanis (1) stated that, treated medically, the mortality was 90 per cent, treated by drainage, it was 40 per cent. In 1934, Brunn (2) reported a mortality rate of 35 per cent in 205 patients, Cutler and Gross (3) a 45 per cent mortality in 47 patients, and in 1940, Sweet (4) reported 34 per cent mortality in 124 cases. Yet as Betts (5) wrote in 1941, "It is rare to find a reported cure rate of 50 per cent or over."

The application of three principles has greatly improved the results in treating lung abscesses. These principles are.

1. Control of infection by antibiotics;
2. Use of postural drainage, based on a knowledge of bronchial anatomy,
3. Earlier surgical treatment—either drainage or resection.

With prompt surgical drainage, in 1942 Neuhof (6, 7) and associates reported a remarkable fall of mortality to 3.27 per cent, and in 1950 they (8) reported 4 operative deaths in 165 cases—an operative mortality rate of 2.43 per cent. Of these, 154 were operated on in the prepenicillin era. Follow up of 115 of these patients for from 5 to 25 years showed 87 per cent free from symptoms, with healed wounds and negative chest films or bronchograms. In 15 cases, however, there were residual postoperative pulmonary cavities or bronchiectasis. Six cases had had hemoptysis, 9 others were symptom free.

Brock (9), however, in a series of 176 cases treated by operation at all stages from acute to late chronic, reported a 26.5 per cent mortality. This, he states, in great part reflects "the poor condition of many chronic neglected cases in which the patients were operated on after failure of prolonged medical treatment."

Although at first sight antibiotics appeared to influence physicians to a return to conservatism, surgical treatment is nevertheless still required, above all for two reasons.

1. There is an increase in the incidence of abscesses due to antibiotic-resistant organisms.
2. Though postural drainage may partially empty an abscess, the cavity often remains as an infected "cyst" filled with inspissated pus and acting as a focus of chronic infection and a source of continuing ill health.



Fig. 1A. Roentgenogram showing pneumonia.



Fig. 1B Pneumonitis advancing to abscess formation.



Fig 1C Stage of endobronchial rupture The abscess fully developed

### PATHOLOGY

A fully-developed lung abscess—a pus-containing cavity within the lung parenchyma—is the final necrotic stage of a suppurative pneumonitis (Fig 1), all degrees of variation from pneumonitis with multiple small abscesses to a large single abscess may occur. Lung abscess is seen at any age. Males are more commonly affected than females, in a ratio of three to one (13).

Like empyema, lung abscess is rarely a primary lesion but is almost always secondary to some detectable cause which should be sought and found before treatment is started. Brock (9) reported over 75 per cent due to identifiable causes and, as Cleland (10) suggests, these fall into *five major* groups

- 1 *From bronchial occlusion* by.
  - (a) Aspiration of septic material causing atelectasis,
  - (b) Bronchial carcinoma (10 to 12 per cent);
  - (c) Benign bronchial tumors,
  - (d) Bronchostenosis,
  - (e) Intrabronchial foreign bodies (1 per cent).



Fig. 2A. Neoplastic lung abscess showing calcification in its wall and in the hilar lymph nodes.

- 2 From specific pneumonias especially
  - (a) Staphylococci,
  - (b) Actinomyces, Friedlander's bacillus,
  - (c) Anaerobic organisms, and rarely from pneumococci and streptococci.
- 3 From nonspecific suppurative pneumonia—*aspiration pneumonia*
- 4 From vascular occlusion by
  - (a) Embolus
  - (b) Pyemia
- 5 From trauma.
  - (a) Producing an infected hematoma
  - (b) Due to a foreign body in the lung

**Pathogenesis.** In his classic studies on lung abscess, Brock (11, 12) has convincingly shown that it is most commonly due to *bronchial embolism* from inhalation of septic material during sleep, narcosis, or anesthesia. The embolus is most commonly derived from septic gums or dental tartar but can be derived from nasal and/or pharyngeal secretions whether mucoid or purulent blood clot after tonsillectomy or



Fig 2B Resected specimen of neoplastic lung abscess seen in Fig 2A

dental extraction, postoperative vomitus or, rarely, bronchiectatic pus. It must never be forgotten that *at least 12 per cent of lung abscesses are due to carcinoma of the lung* (Maxwell (13), 10·8 per cent, Brock (11), 13·8 per cent) (Figs 2A and B).

**Bacteriology.** Whereas the *acute fetid abscesses* are usually due to mixed infection of spirochetes, fusiform bacilli, and anaerobic streptococci, the nonfetid abscesses are due to staphylococci, streptococci, and Friedlander's bacillus

Of the specific lung infections, staphylococcal pneumonia is not infrequently complicated by the formation of small, multiple, subpleural abscesses, and, especially in children, one of these may rupture into the pleural cavity producing tension pyopneumothorax (Chapter 9, Fig 4).

## Pathology

**Location.** Abscesses due to bronchial embolism always form *peripherally* in a dependent lung segment. The bronchus affected is determined by whether the patient is lying on his side, front, or back at the time of bronchial embolism. The posterior segment of the right upper lobe is most commonly affected, followed by the apical segments of the right and left lower lobes (Fig 3). The aim of clinical investigation, therefore, is to decide which anatomic lung segment is affected, for this forms the basis of logical treatment (Chapter 1, Fig 1).

**Development of the Abscess.** Occlusion of a bronchus is rapidly followed by segmental or subsegmental atelectasis, stasis, and suppurative pneumonitis whose severity varies with the virulence of the infecting organisms. Within 10 to 14 days gangrene, sloughing, and cavitation will have occurred in the affected segment (Fig 1). As the abscess develops near the surface of the lung, there is invariably an overlying pleurisy with adhesion formation and local fusion of the pleural layers. "Surgically speaking, it therefore becomes a matter of deciding where the adhesions exist, not if they exist" (14). Rarely a fulminating abscess may rupture into the pleural cavity before any adhesions have formed and may thus cause pyopneumothorax.

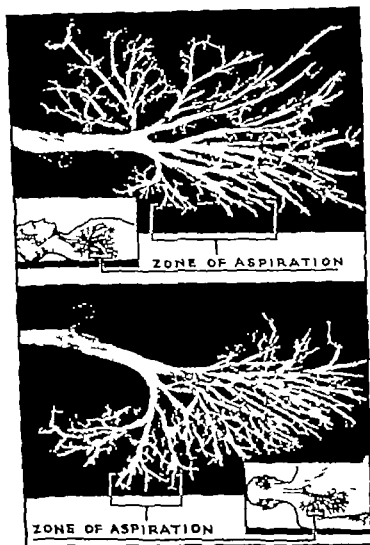


Fig 3 Pathogenesis of lung abscess. A, With patient supine, bronchial emboli gravitate into dependent apical bronchus of lower lobe. B, When patient lies on side, most dependent bronchi are axillary divisions of anterior and posterior segments of upper lobe. (Courtesy Lindskog, G. E., and Liebow. A. A. Thoracic Surgery and Related Pathology 1953 p 144 fig. 71.)



Brock found that all his 117 cases of abscess treated by operation were superficial 103 faced the thoracic cage, 6 faced the fissures, 5 faced the vertebrae, 2 faced the mediastinum, and 1 faced the diaphragm None lay deeply.

At first, the intrabronchial communication is occluded by swelling, edema, and tension Sooner or later, the abscess discharges into a bronchus, either intermittently or as a "sudden massive emptying."

This fully developed abscess may present in one of three forms

1. Filled with a large slough of surrounding lung (Fig 4),
- 2 Without slough;
- 3 As a chronic pneumonitis.

In the presence of slough, rapid effective intrabronchial drainage is impossible, and surgical drainage is the only logical treatment.

When the drainage has been better and the slough minimal, complete emptying, followed by rapid healing of the abscess without permanent sequelae, can occur (Figs 5A-C)



Fig 4 Large slough in acute lung abscess secondary to fractured jaw

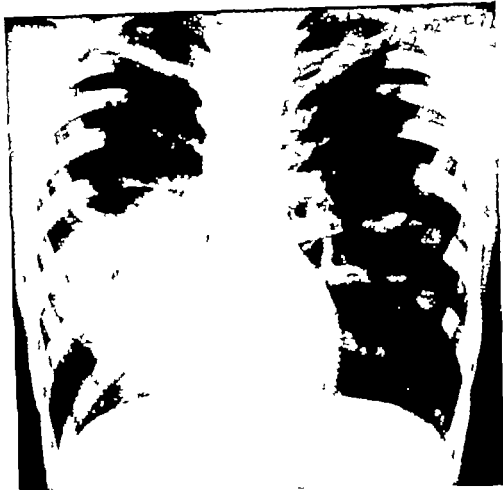


Fig. 5A. Posteroanterior view of abscess in right lung.



Fig. 5B Lateral film confirming abscess in apical segment of right lower lobe.



Fig 5C Resolution without sequelae after bronchoscopic aspiration and posteral drainage, postoperative treatment bronchogram showing minimal bronchiectatic change in apical segment (Anterior and middle segments have not filled )

Finally, when neglected, a lung abscess may persist as a partially collapsed, thick-walled abscess surrounded by dense fibrous tissue and filled with semisolid granulations. It cannot heal unaided but remains as a source of further emboli and additional lung abscesses (Fig 6).

**Complications of Lung Abscess.** Despite chemotherapy, complications do still occur, and an apparent clinical response of the abscess to “medical treatment” should not blind us to this very real possibility. The complications may be either local or general.

**LOCAL COMPLICATIONS** As already indicated, the abscess may spread directly into adjacent lung segments or across a fissure. It may also spread by bronchial embolism to form fresh abscesses. Pleural effusion or empyema may occur without intrapleural rupture, or sudden pleural rupture may precipitate a tension pyopneumothorax.

**GENERAL COMPLICATIONS** These occur as.

1. *Cerebral abscess* which may be single or multiple and which arises either by systemic blood stream spread or via the vertebral venous plexus (15, 16);
2. *General dissemination* The patient may develop multiple abscesses, generalized arthritis, and a profound and rapidly advancing anemia.

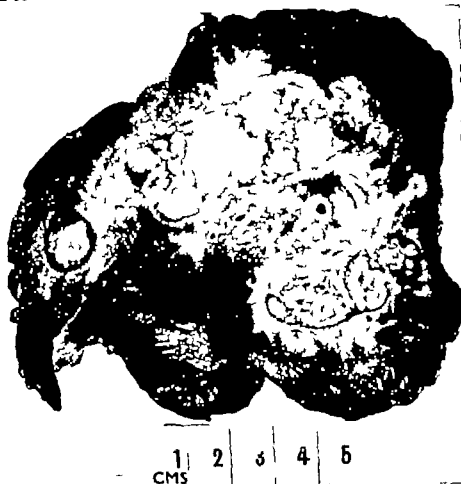


Fig 6. Chronic lung abscess, surrounded by dense fibrous tissue and filled with semisolid granulations; resection specimen.

### CLINICAL FEATURES

Clinical features vary with the cause and severity of the infection, and they mirror the pathologic progress. They will be considered as they present with

- 1 The acute abscess
- 2 The subacute abscess.

**The Acute Abscess.** Following an initial latent period of 10 to 20 days during which the causal factor is acting, an acute lung abscess typically has the sudden onset of a severe febrile illness associated with pleural pain and generalized toxemia. The patient rapidly loses weight and becomes anemic, toxic, and gravely ill. When arising after a surgical operation, the acute abscess may masquerade as postoperative bronchitis with fever, cough, sputum, pleural pain, and general malaise.

Some 7 to 10 days after the onset of severe symptoms—and often preceded by a warning foetor and hemotysis—the developing acute abscess ruptures into the bronchial tree. The patient coughs up copious foul-smelling brownish sputum which, if collected into a tall glass, can separate into three typical layers—froth, fluid, and solid. On other occasions the sputum may appear gradually and be preceded by hemoptysis. If endobronchial drainage of this abscess is adequate, symptoms can regress rapidly; but if not, the abscess may spread, form embolic abscesses throughout both lungs, and cause the complications already mentioned, leading to toxic arthritis, nephritis, and terminal bronchopneumonia.

**The Subacute Abscess.** A subacute abscess, or one masked by antibiotics, develops insidiously and is often detected only when chronic ill health, pleural pain, cough, and sputum demand full investigation. There are alternating periods of relief and recurrence of symptoms with fever, increased sputum, and a steady decline in health.

Although a neoplastic abscess is usually subacute or chronic in onset, the author has encountered it as an acute fulminating lesion advancing to pyothorax.

**Physical Signs. GENERAL.** With an *acute abscess*, the foul smell as well as the appearance of the patient sitting up, continually coughing, freely expectorating, and anxiously clutching a half-filled sputum pot is typical. When combined with septic gums, dental caries, or a history of a recent surgical operation and postoperative "bronchitis," the diagnosis is certain. The patient has pale, sunken cheeks and an anxious expression. Frequently, there is clubbing of the fingers and toes.

With a *subacute abscess*, there is much less cough and sputum and less severe signs of general ill health.

**LOCAL.** These vary greatly from few signs to those of segmental collapse of the lung. When a pleural effusion has developed, there are signs of fluid. When intrapleural rupture has occurred, the signs of pyopneumothorax are present and are confirmed by thoracentesis.

**Investigations.** There are three important investigations:

- 1 Sputum examination,
- 2 Roentgenograms of the chest;
- 3 Bronchoscopy.

**SPUTUM EXAMINATION.** With an acute abscess, the smell, color, and volume of sputum is characteristic. It is examined for acid-fast bacilli and malignant cells and cultured aerobically and anaerobically for pathogens, especially staphylococci and Friedlander's bacillus. The volume of sputum expectorated each day is measured and graphed.

**ROENTGENOGRAMS.** Two-plane roentgenograms are required. Not only do they accurately localize the abscess, but when taken serially they are the most valuable single guide to the progress of the abscess. The findings vary with the stage of the abscess.

In the *earliest phase*, there is lobar or segmental atelectasis or consolidation (Fig 1). As the abscess develops, the shadow tends to become more spherical, but, because of surrounding pneumonitis, the outline remains hazy. With endobronchial rupture, a fluid level will usually be seen; and, depending on the efficacy of treatment, the level in serial films may rise or fall. When a slough partially occludes the bronchial opening, its shape is outlined by surrounding air. With total occlusion, no air enters and no fluid level is seen. Following intrapleural rupture, a fluid level is seen in the pleural cavity.

In the *hyperacute* variety of abscess, Neuhoff (7) emphasized four typical features:

- 1 A large area containing a
  - 2 More infiltration of the abs
  3. An irregular order due to
  - monary slough
  4. An intense and reaction
- in an acute abscess;  
grenous pul-



Fig 7A. Lung abscess treated medically (Jan. 16 1956)

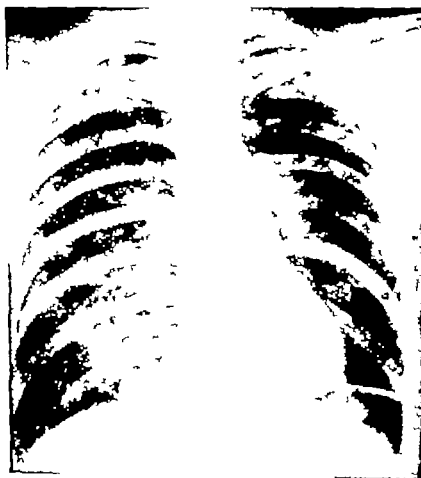


Fig 7B Despite postural drainage one year later abscess remained as thin walled cyst (Jan. 17 1957) causing chronic invalidity



Fig 7C Resected specimen showing epithelialized lining

When postural drainage treatment is successful, the abscess usually shrinks in size over the next two to three weeks, and the surrounding zone of pneumonitis gradually fades (Fig 5C). At times, however, though the abscess is drained, the cavity wall does not collapse but remains as a thin-walled cyst (Figs 7A and B).

In subacute abscesses, further roentgenograms, including lateral tomography or bronchography, are required. Bronchography is also advised when treatment is completed, to exclude residual bronchiectasis.

**BRONCHOSCOPY** This is an essential step in the management of every lung abscess. It may reveal the cause, such as carcinoma or foreign body, and it can be the first step in effective treatment (Fig 5C).

**Differential Diagnosis.** A developing lung abscess simulates lobar pneumonia. When established, it must be differentiated from an empyema (especially with bronchopleural fistula), carcinoma of the lung, infected lung cyst (including hydatid cyst), and a tuberculous cavity.

**Empyema.** A lung abscess may simulate empyema thoracis, and, if either has caused a bronchopleural fistula, further differentiation may be impossible. In each of these conditions, sputum is considerable, there is secondary contraction of the chest wall, roentgenologically the shadowing extends to the costophrenic sinus, and aspiration will reveal pus.

**Carcinoma of the Lung** A peripheral carcinoma of the lung with cavitation and fluid level can often be distinguished radiographically by its thick, irregular outline (Fig 8). Regular walls and diminution in size of the cavity by postural drainage, however, are no guarantee that the lesion is not neoplastic, nor does calcium in the walls of the cavity denote only a tuberculous etiology (Fig 2). Any chronic peripheral abscess, especially in a patient over 40 years of age, should be viewed with the utmost distrust and should be regarded as neoplastic until proved otherwise.

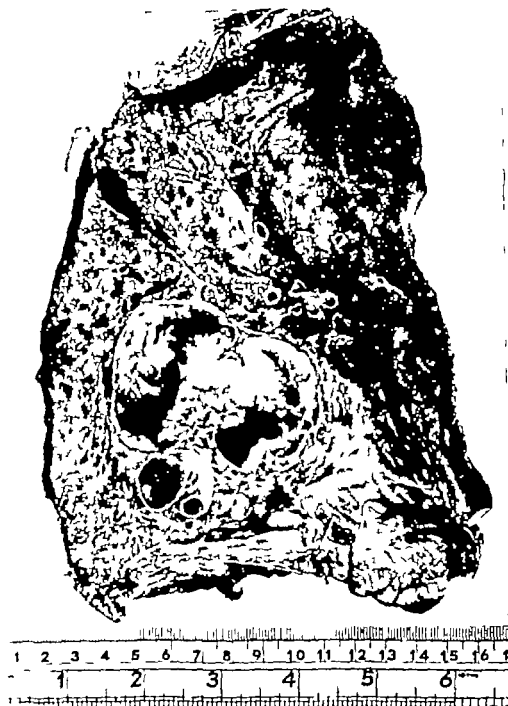


Fig 8. Thick walled neoplastic lung abscess.

*Infected Lung Cysts* These cysts are spherical in shape, and when communicating with a bronchus have a fluid level that varies in height. From time to time surrounding pneumonitis is also present.

In sheep-rearing countries such as New Zealand, infected *hydatid* cysts simulate acute lung abscess in all their characteristics. Specific tests such as the hydatid complement fixation and Casoni tests are of value. Eosinophilia is confirmatory. When the cysts have ruptured into a bronchus and the fluid has been coughed up roentgenograms are characteristic (Fig 9).

*Tuberculous Cavity* This specific "lung abscess" is differentiated by sputum findings, site and chest roentgenograms. Whether an abscess is tuberculous or not can sometimes be decided only by histologic examination after excision (Fig. 10)



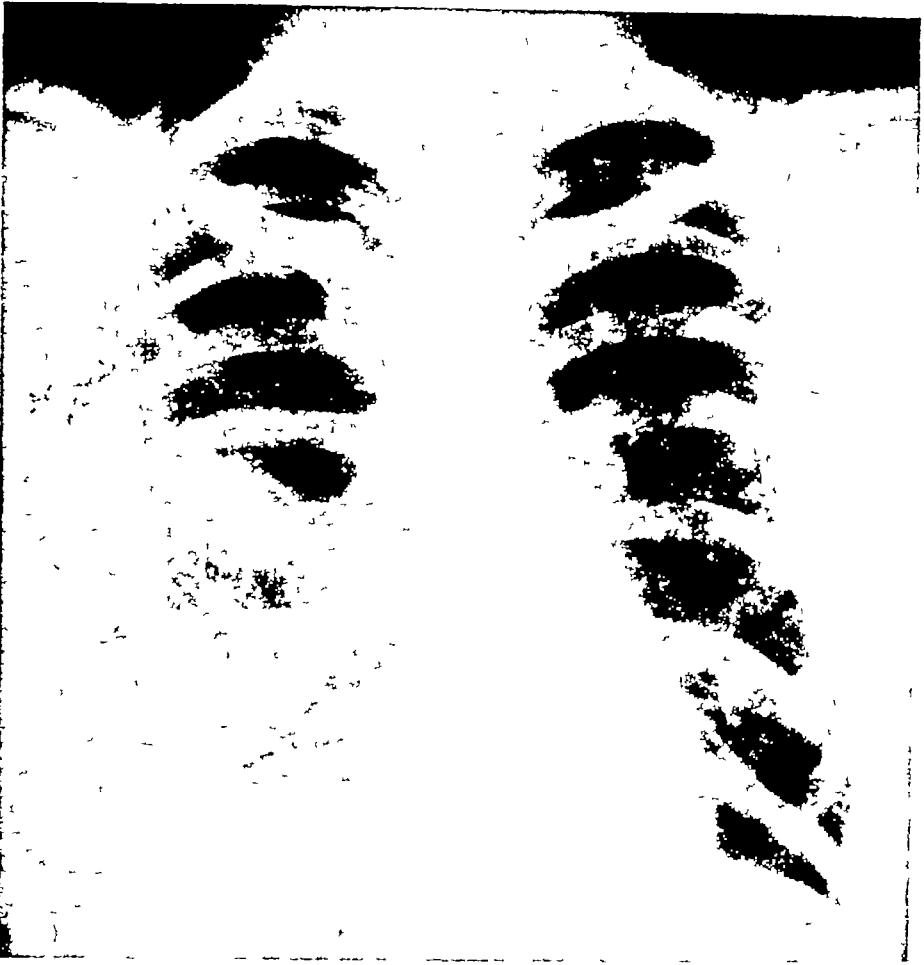


Fig 9 Ruptured infected hydatid cyst, with crumpled endocyst, lobectomy successful

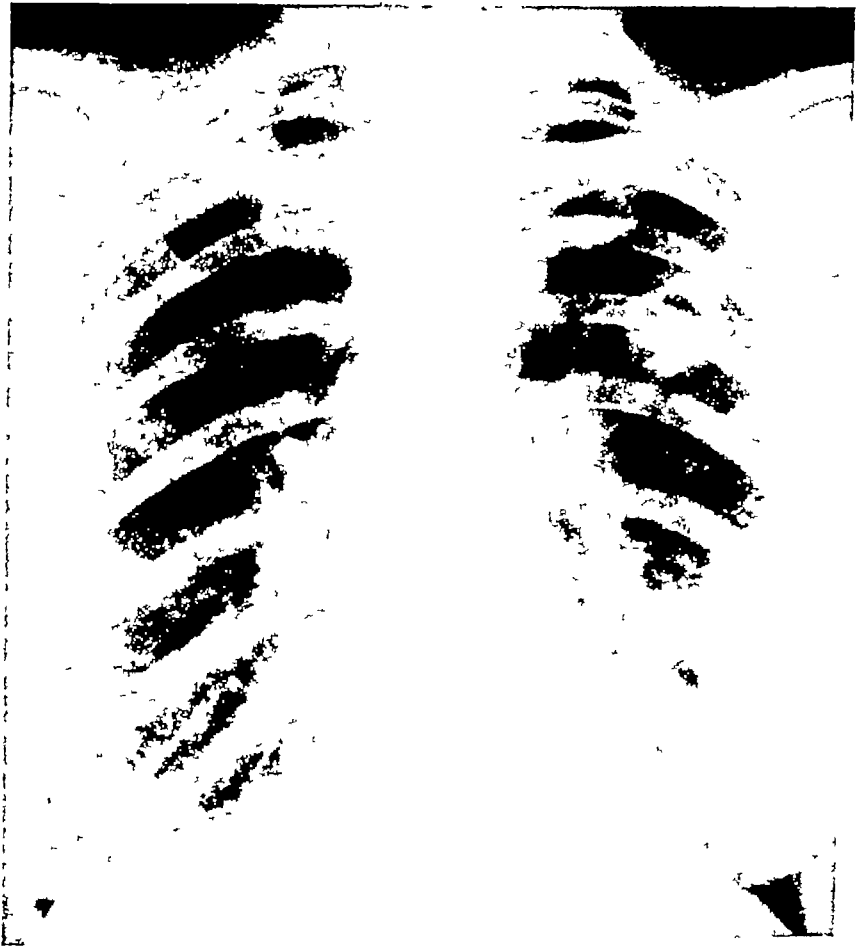


Fig 10 Peripheral lung abscess in left lung—regarded as tuberculous, proven chronic pyogenic only after resection

## Principles of Management

**Prognosis.** All lung abscesses are serious, and though some, especially in young, may resolve completely many leave residual sequelae. There are grounds for complacency in their management, for, even though the abscess is regressing or have regressed, complications can still occur.

In summary therefore, the essential steps of investigation are to

- 1 Check the site by roentgenograms,
- 2 Check the organism by sputum culture,
- 3 Check the cause by bronchoscopy

### PRINCIPLES OF MANAGEMENT

Brock (9) epitomizes the principles of management as

- 1 To encourage resolution or maturation and spontaneous discharge, rest, chemotherapy, and general nursing;
- 2 After endobronchial rupture, to encourage emptying of the abscess and resolution by bronchoscopic and postural drainage
- 3 If the abscess is enlarging, to incise,
- 4 If the abscess is stationary and is suspected of being a cavity

The management therefore varies with the stage at which the abscess is found.

#### EARLY STAGE

With an acute inflammatory process in the lung, the lung is treated with antibiotics. Penicillin, 1 million units twice daily, is given. The organism in the sputum is verified thereafter the appropriate treatment administered. Bronchoscopy is required to check whether or not body bronchial occlusion is present. The progress of the patient is followed by roentgenograms repeated at two-day intervals. If the patient is responsive the shadowing will lessen. The shadowing may however extend and enter either the stage of endobronchial rupture or the fulminating stage.

#### STAGE OF ENDOBRONCHIAL RUPTURE

As already mentioned, endobronchial rupture may be accompanied by copious foul-smelling sputum or quietly with a significant amount of blood. Bronchoscopy is required. To avoid the possibility of the abscess being missed, bronchoscopy is performed with a minimal amount of local anesthesia. The patient is placed head down. When the abscess lies in the apical segment, it is frequently possible to pass the soft rubber aspirating catheter into this bronchus and into the abscess cavity. The suction is applied and a yellow pus immediately appears in the Luken tube. A comes tinged with blood. (This procedure should not be attempted if there is little or no chance of entering the abscess cavity of an upper lobe.) (Figs. 5A-C)

Thereafter the bronchoscope is removed and the patient is placed on his side for postural drainage with bronchoscopic drainage also palliates the condition.

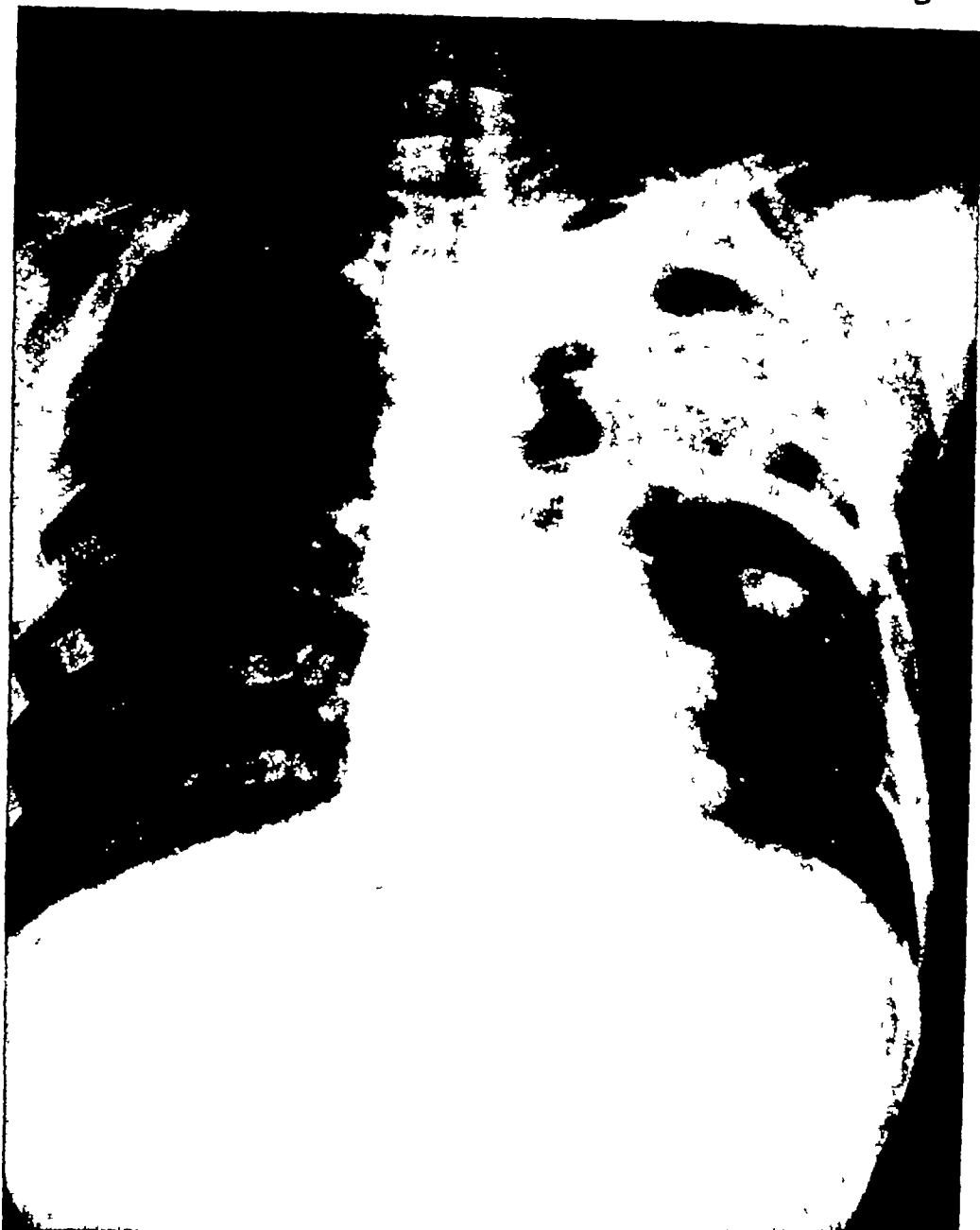


Fig 11A Fulminating left upper-lobe abscess associated with profound clinical deterioration despite medical treatment, emergency drainage

**Physiotherapy.** Continuous postural drainage together with repeated vibration therapy, at least three times daily, until the quantity of sputum lessens, greatly enhances the chance of a complete cure. Thereafter, breathing exercises ensure complete re-expansion of the lung.

**Antibiotic Therapy.** Antibiotic therapy, started in the early stage, is continued

#### EVALUATION OF MEDICAL TREATMENT

With medical treatment, one of three things may happen

1. If the fever abates, the sputum lessens, and the serial roentgenograms show progressive improvement, it is likely that the abscess will resolve. Final bronchograms—a month after complete resolution—are required to check that there is no residual bronchiectasis (Fig 5C)
2. There may be delayed resolution (Fig 4),



Fig. 11B Pack in drained abscess, persisting sinus.

- 3 The patient may continue to be profoundly ill and may rapidly become emaciated with hectic temperatures and increasing size of the abscess. This indicates a fulminating abscess that requires prompt rib resection and external drainage if life is to be saved. At a later date the destroyed lung segment may be resected (Figs 11A-D)

#### FULMINATING STAGE

**Operative Treatment.** Even in 1924 Romanis (1) advised that once the diagnosis is made and the decision taken to operate it should not be delayed beyond 24 hours for further localizing roentgenograms to be made

**WHERE TO DRAIN** The site for drainage is selected either by studying two-plane roentgenograms, carefully counting ribs on the patient, or preferably by fluoroscopy when the overlying skin is marked with a skin pencil

**ANESTHESIA.** Local anesthesia, as for empyema, is desirable as inhalation anesthesia may precipitate drowning

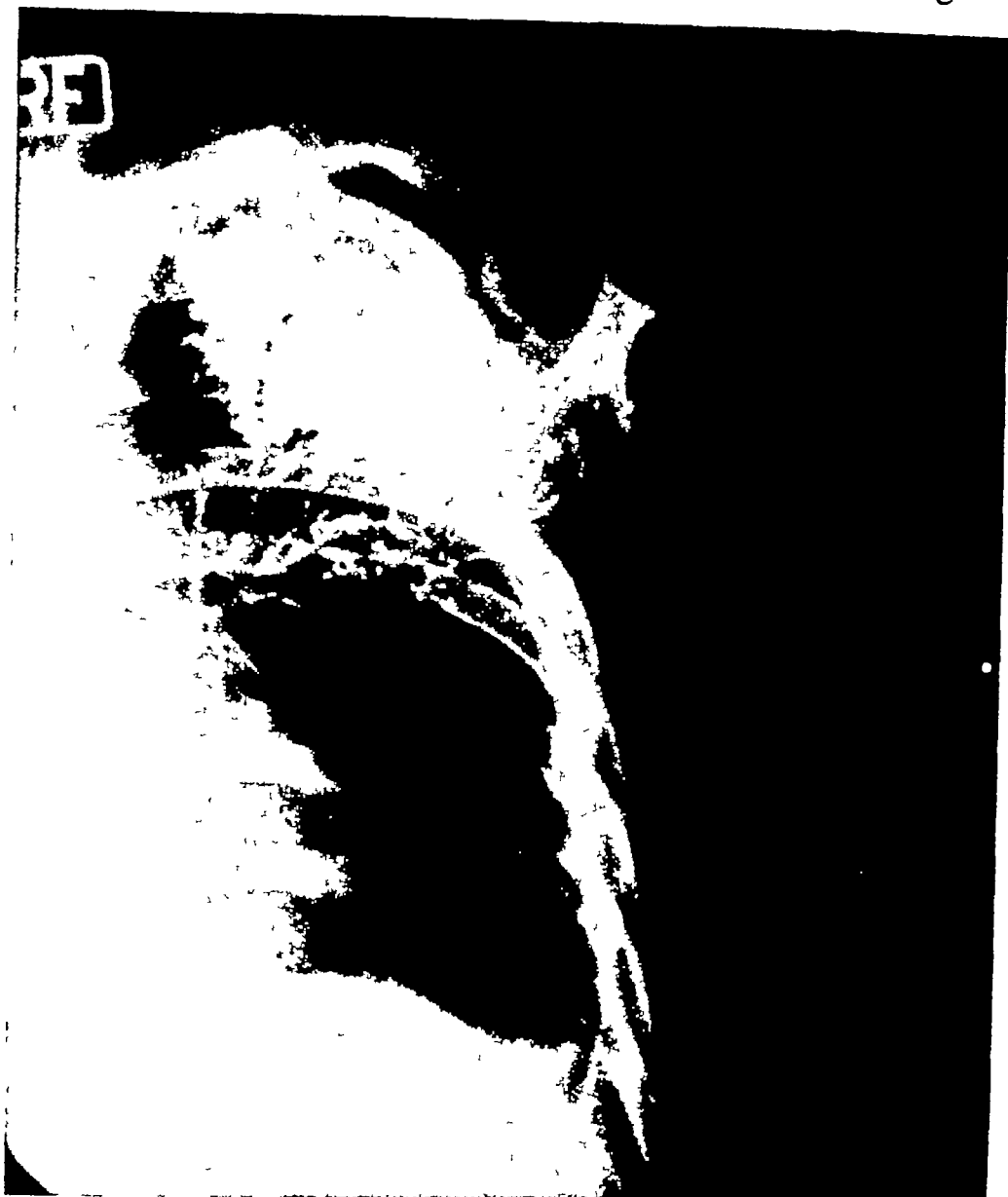


Fig 11C Sinogram reveals destroyed upper lobe and uncollapsed abscess cavity

**POSITION FOR SURGICAL DRAINAGE** This depends on the site of the abscess. If the abscess is in the axilla, the patient reclines on the opposite side with the head and shoulders raised, if it is in the pectoral segment, he reclines on his back, if it is in the apical segment of the upper or lower lobes, he lies prone with head down.

**TECHNIC** (Fig 12). The technic of operation is essentially the same as that for empyema. A 6 cm vertical incision is made over the rib, the muscular layer is divided, and the periosteum is isolated, incised, and reflected over 4 to 5 cm. The associated intercostal vessels are ligated at either end and the segment of rib removed, together with the neighboring intercostal muscle.

Palpation discloses if the underlying lung is indurated, and aspiration of pus and/or air confirms the depth and the direction for further incision. Drainage is usually performed in *one stage*.

When, however, there is no induration and no adhesion formation and the underlying lung moves freely, a further check of position is required. If the site proves correct, then *two-stage drainage* is required. The operation is stopped, a swab soaked in iodine solution is left in against the parietal pleura to encourage adhesion forma-



Fig. 11D Bronchogram after successful left upper lobe resection. (Courtesy George A. Mason.)

tion, and the skin edges are approximated. A week later the second stage is completed.

In the second operation, the lung parenchyma is divided, preferably with cutting diathermy or cautery and the abscess cavity is opened. Pus is sucked clear, casts and slough are removed, and the cavity is inspected before enlarging the opening. The parietal wall is removed, thus unroofing the cavity, and any loculi are broken down to establish free drainage. If the cavity extends beneath a rib a further segment should be excised together with the intercostal bundle.

The cavity is next packed with petrolatum gauze. Mason (17) prefers Rutherford Morrison's B.I.P.P.\* gauze. The author has found this to be satisfactory and it has the added advantage of being radiopaque (Fig. 11B). A pad held firmly with elastoplast completes the dressing.

\*B.I.P.P. is Bismuth Iodoform Paraffin Paste.

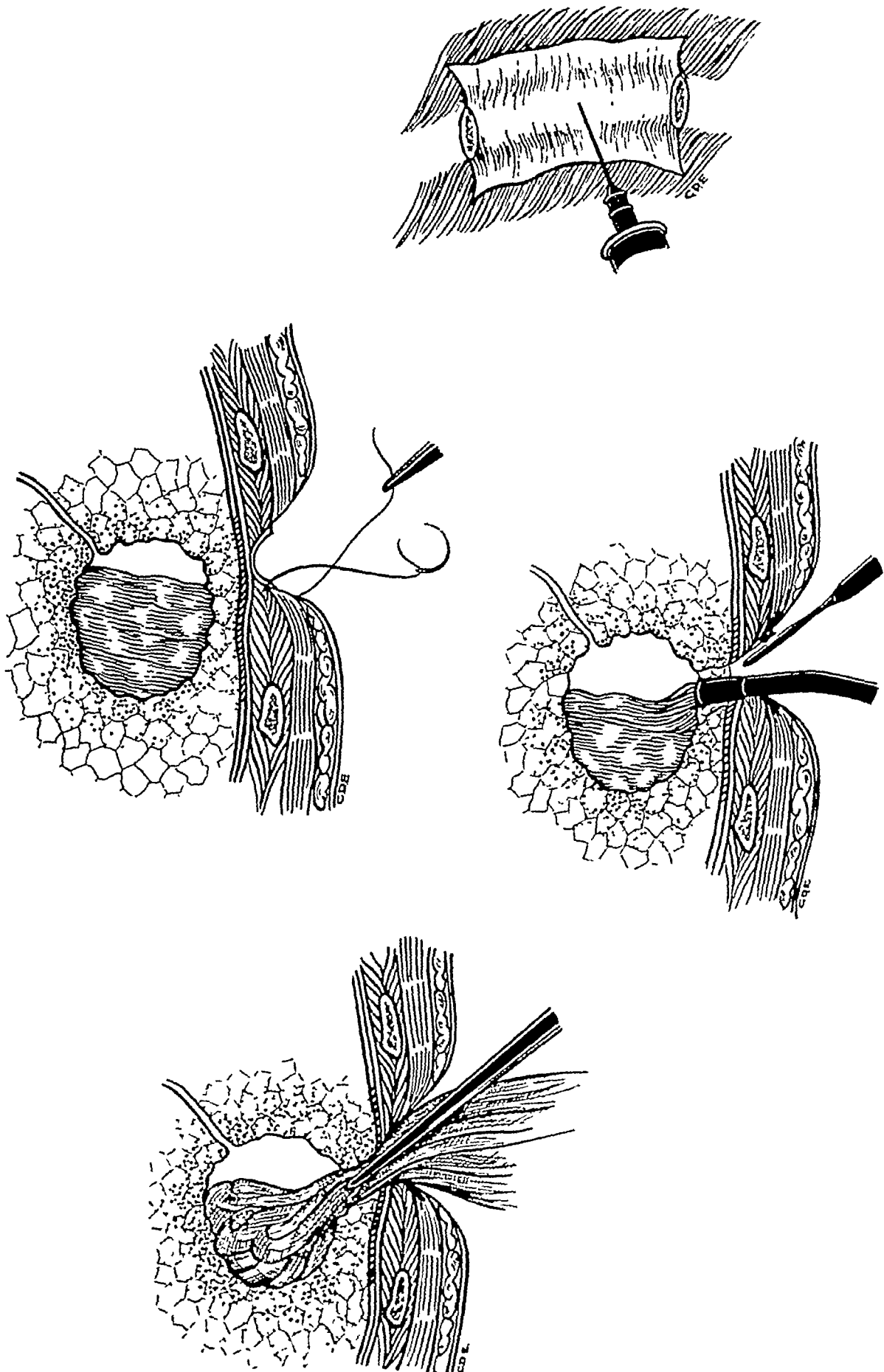


Fig. 12 Technic for drainage of lung abscess 1, Following rib-resection, the abscess is finally located by aspiration 2, Periosteum and subcutaneous layers are approximated with sutures 3, The abscess cavity is opened with a cautery 4, The abscess is finally packed with gauze, and dressings are applied. (See page 139.)

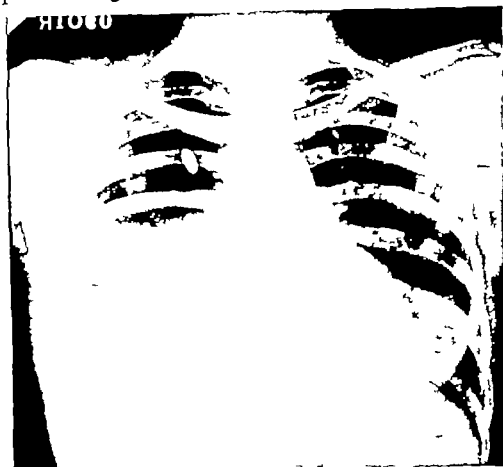


Fig. 13A. Right pyopneumothorax from ruptured lower-lobe hydatid cyst, showing characteristic dome shape of endocyst in lower corner

When the line of adhesion is narrow and the pleura has been opened inadvertently it is sutured and reinforced with an intercostal muscle bundle. Because an airtight seal may be difficult to achieve, water-seal drainage is inserted at the base of the pleural cavity and air removed by continuous aspiration. This not only avoids a possible empyema but also, by keeping the lung fully expanded, seals any residual leak.

**Postoperative Management.** *The pack is changed in four or five days.* Because of the inevitable bronchopleural fistula, irrigation is forbidden. When the cavity narrows to a sinus track, a tube is inserted to keep the skin from closing in before the depths are healed. Hemoglobin is checked at weekly intervals. If it becomes 70 per cent or less, a blood transfusion is given.

*Chemotherapy* is continued until the patient becomes afebrile, iron therapy until blood counts are normal, and *deep breathing exercises* until chest roentgenograms and ventilatory function are normal. The patient is also encouraged to eat all he can of a high protein diet.

*Secondary hemorrhage* can occur. It may be sudden and severe, arising either from an intercostal or a pulmonary arterial branch. Emergency packing of the cavity and firm elastoplast binding must be followed by careful inspection in the operating room. If the bleeding point is an intercostal artery with its higher systemic pressure, the bleeding point must be secured and sutured. If bleeding is from a branch of the pulmonary artery with its lower pressure, a blood clot will probably already



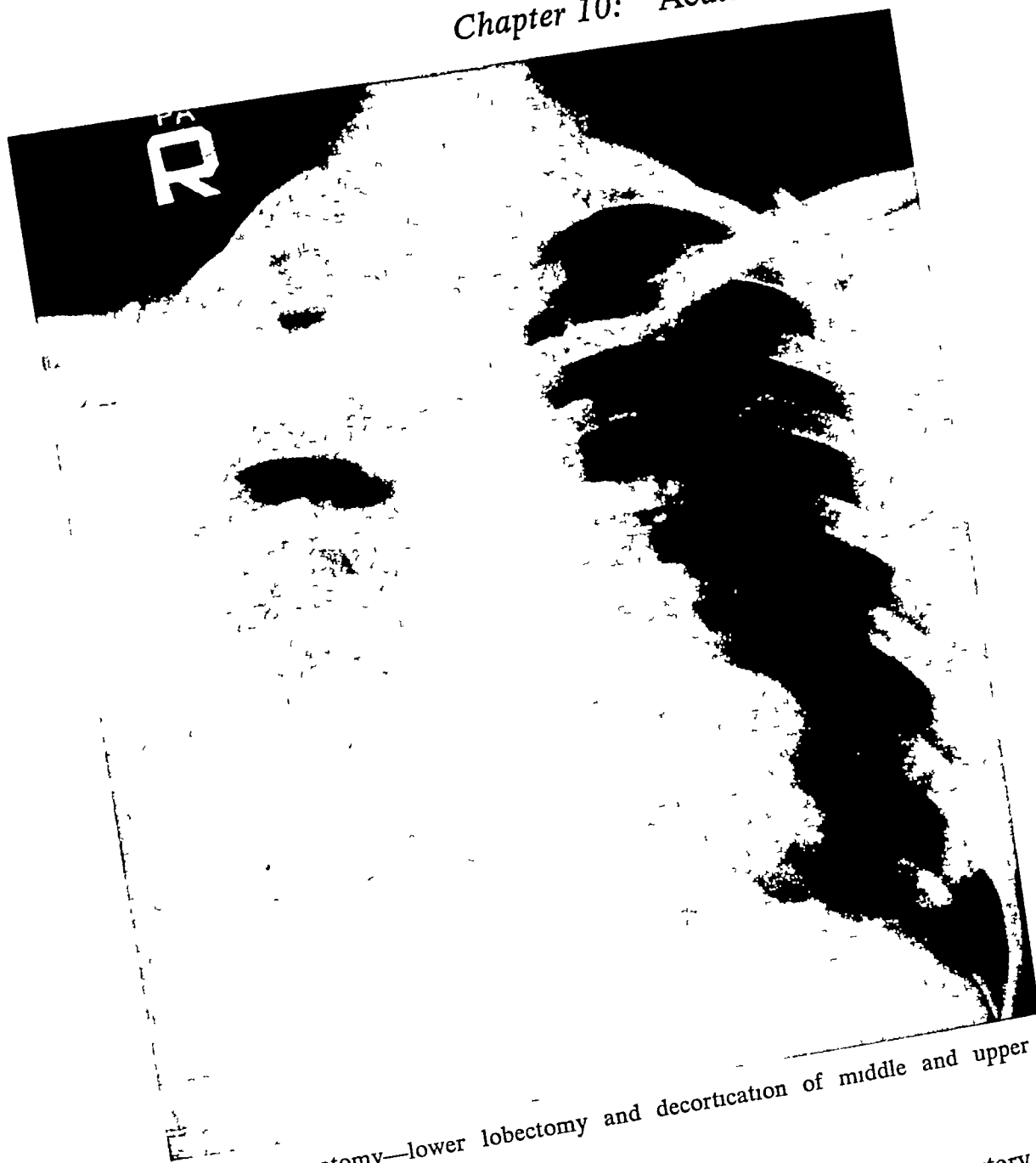


Fig 13B Thoracotomy—lower lobectomy and decortication of middle and upper lobes  
(Courtesy Douglas Robb)

formed. If bleeding continues, however, the point in the pulmonary artery should be similarly secured.

**Resection.** The *indications* for resection are

- 1 Delayed resolution,
- 2 Severe hemoptysis,
- 3 Infected cysts which are in fact abscesses—e g an infected hydatid cyst,
- 4 Specific lung abscesses

**Delayed Resolution** This arises when, despite internal or even external drainage, there are frequent clinical relapses and no convincing roentgenologic change in the appearance of the abscess. After preliminary bronchography to check the remaining lung, and sinography—if a sinus is present (Fig 11C)—resection is required.

**Massive Hemoptysis** In massive hemoptysis from a chronic abscess, preliminary bronchoscopy is performed (a) to confirm the source of the bleeding, and (b) to

plug the draining bronchus tightly with a swab or anesthetic bronchial blocker before proceeding to immediate resection.

*Infected Cyst—Hydatid Cyst* With endobronchial rupture a hydatid cyst in variably becomes infected. If the cyst is large, the greater part of the affected lobe will have been destroyed and lobectomy is clearly indicated (Fig 9). However, when the cyst is small, segmental resection is advised.

When a hydatid cyst has ruptured into the pleural cavity and caused pyopneumothorax, the patient must be saved from drowning by intercostal water-seal drainage. Thereafter, a planned thoracotomy is required together with resection of the destroyed cyst-containing lobe and decortication of the remaining lobes (Figs 13A and B).

*Specific Lung Abscesses* Other specific lung abscesses such as pulmonary histoplasmosis (18) and coccidioidomycosis (19, 20) require resection, but these resections are beyond the scope of this book. The reader is referred to Hodgson (18), Melick (19), Cotton (20) and their associates for excellent summaries of contemporary experience.

#### REFERENCES

- 1 Romanis, W. H. C. On the treatment of empyema and acute abscess of lung, *Practitioner* 113 33 1924
- 2 Brunn, H. Lung abscess, *J.A.M.A.*, 103 1999 1934
- 3 Cutler E. C., and Gross, R. E. Nontuberculous abscess of the lung; etiology, treatment, and results in 90 cases, *J Thoracic Surg.*, 6 125 1936.
- 4 Sweet, R. H. Lung abscess: analysis of Massachusetts General Hospital cases from 1933 through 1937. *Surg. Gynec. & Obst.*, 70 1011 1940
- 5 Betts, R. H. Principles in the management of pulmonary abscess, *Am. J Surg.*, 54 82, 1941
- 6 Neuhoef H., and Touroff, A. S. W. Acute putrid abscess of lung. IV. Surgical treatment and results of 36 consecutive cases, *J Thoracic Surg.*, 9 439 1940.
- 7 ——— and Touroff A. S. W. Acute putrid abscess of lung. V. Hyperacute variety. *J Thoracic Surg.* 12 98 1942.
- 8 Touroff A. S. W., Nabatoff, R. A., and Neuhoef H. Acute putrid abscess of the lung. VI. The late results of surgical treatment, *J Thoracic Surg.* 20 266 1950
- 9 Brock, R. C. "Pulmonary Abscess" in *British Surgical Practice*, 1st ed., London, Butterworth & Co., 1950 7 185
- 10 Cleland, W. P. "Lung Abscess" in *Diseases of the Chest* edited by Marshall, Sir G. and Perry K. M. A. 1st ed., London, Butterworth & Co., 1952, 1 197
- 11 Brock, R. C. Studies in lung abscess, *Guy's Hosp. Rep.*, 97 75 1948.
- 12 ——— Lung Abscess, 1st ed. Oxford, Blackwell Scientific Publications 1952.
- 13 Maxwell, J. Lung abscess, with special reference to causation and treatment, *Quart. J Med.*, 3 467 1934
- 14 Neuhoef H., Touroff A. S. W., and Aufses, A. H. The surgical treatment by drainage of subacute and chronic putrid abscess of lung, *Ann. Surg.*, 113 209 1941
- 15 Batson, O. V. The function of the vertebral veins and their role in the spread of metastases. *Ann. Surg.* 112 138 1940
- 16 Collis, J. L. The etiology of cerebral abscess as a complication of thoracic disease, *J Thoracic Surg.* 13 445 1944
- 17 Mason, G. A. Personal communication.
- 18 Hodgson, E. H., Weed, L. A., and Clagett, O. T. Pulmonary histoplasmosis. Review of published cases and Report of an unusual case, *J Thoracic Surg.*, 20 97 1950
- 19 Melick, D. W. Excisional surgery in pulmonary coccidioidomycosis, *J Thoracic Surg.*, 20 66 1950
- 20 Cotton, B. H., and Birsner J. W. Surgical treatment in pulmonary coccidioidomycosis. Preliminary report of thirty cases, *J Thoracic Surg.*, 20 429 1950

## MASSIVE HEMOPTYSIS

**Introduction.** Hemoptysis is a symptom of pulmonary or cardiac disorder. It varies in severity from a faint staining of the sputum to a sudden and massive hemorrhage. When massive, it is as alarming for the physician as it is dangerous for the patient.

Historically, until the late forties, even massive hemoptysis was regarded essentially as a medical problem. The obvious dangers, however, of profound blood loss, drowning in blood, or fatal spread of an existing tuberculous infection have more recently brought a change to the more logical attitude of promptly arresting the hemorrhage by surgical means.

In 1948, Ryan and Lineberry (1) first performed emergency pneumonectomy for uncontrollable hemoptysis from a tuberculous patient who had coughed up 720 ml of blood in a 24-hour period. In 1952, Ross (2) reported a similar case.

Ehrenhaft and Taber (3), describing the management of 12 patients with massive hemoptysis from causes other than pulmonary tuberculosis, stressed that it constitutes a clear indication for thoracotomy as soon as the bleeding site is localized.

### PATHOLOGY

Massive hemoptysis is rarely, if ever, seen in children. The most common cause is pulmonary tuberculosis. Bleeding can arise from tuberculous ulceration of the bronchial mucosa (rarely severe), from rupture of a Rasmussen type of aneurysm (4) in the medial wall of a tuberculous cavity, or from a calcified hilar node ulcerating into the lumen of a bronchus by pressure necrosis (5). Bleeding may therefore arise from either a pulmonary or a bronchial artery.

The next most common cause is chronic lung sepsis, especially due to bronchiectasis, broncholithiasis, or chronic lung abscess. In this group, bleeding usually comes from a branch of the bronchial artery. Massive hemoptysis from a pulmonary or esophageal neoplasm or aortic aneurysm is usually a terminal event. Death may occur in one of three ways:

1. Blood loss,
2. Drowning in blood,
3. Spread of an existing tuberculosis.

Especially when the patients are nontuberculous, they often appear to be in reasonable health until the episode of massive hemoptysis. Occasionally there are small warning hemorrhages, as in a third of Ehrenhaft's (3) series.

The severity of the hemoptysis bears no relation to the gravity of the underlying pulmonary lesion (3, 6, 7) (Fig. 1). It is the hemoptysis per se and not its cause that threatens life. In three of the author's cases (Figs. 1 through 3), one had a minimal tuberculous lesion which bled and caused bilateral spread of the lesion before it could be referred for a surgical opinion, the second had bronchiectasis requiring urgent lobectomy, the third and fatal case had a minimal apical tuberculous lesion.



Fig. 1 Minimal apical tuberculous lesion that caused fatal hemoptysis.

Ehrenhaft (3) reported two patients with mild bronchiectasis six with broncho-lithiasis or calcified peribronchial lymph nodes, and four with no specific cause for the massive bleeding.

Pathologic examination of the resected pulmonary tissue while revealing the cause rarely demonstrates the exact bleeding point or vessel. Even injection studies, serial sections or examination of the bronchial walls under a dissecting microscope reveal little. Roentgenograms of the specimens merely demonstrate peribronchial calcification.

The remaining lung tissue secondarily develops areas of atelectasis from endobronchial aspiration of blood clot and spread of a tuberculous infection when present.

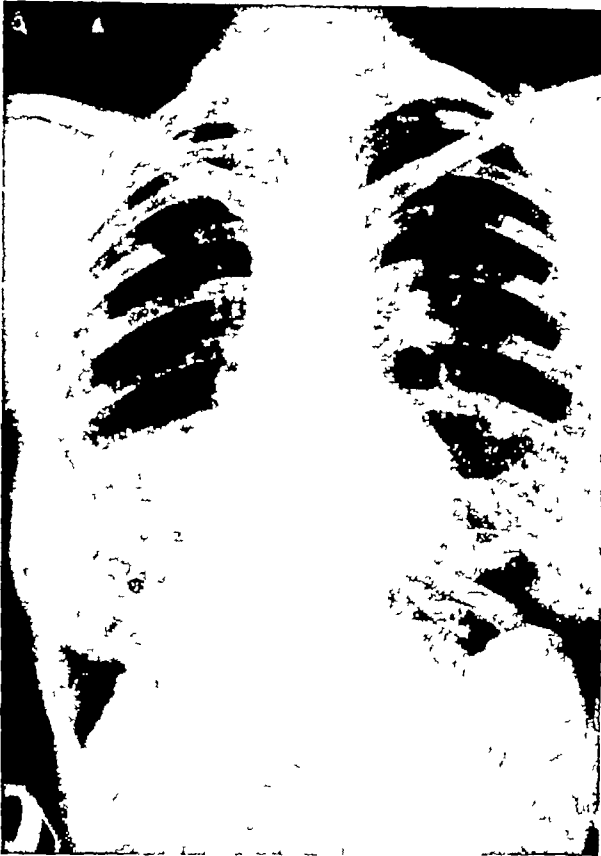


Fig 2A Chest film (Jan 21, 1954) showing collapsed right lower lobe from tuberculous bronchiectasis

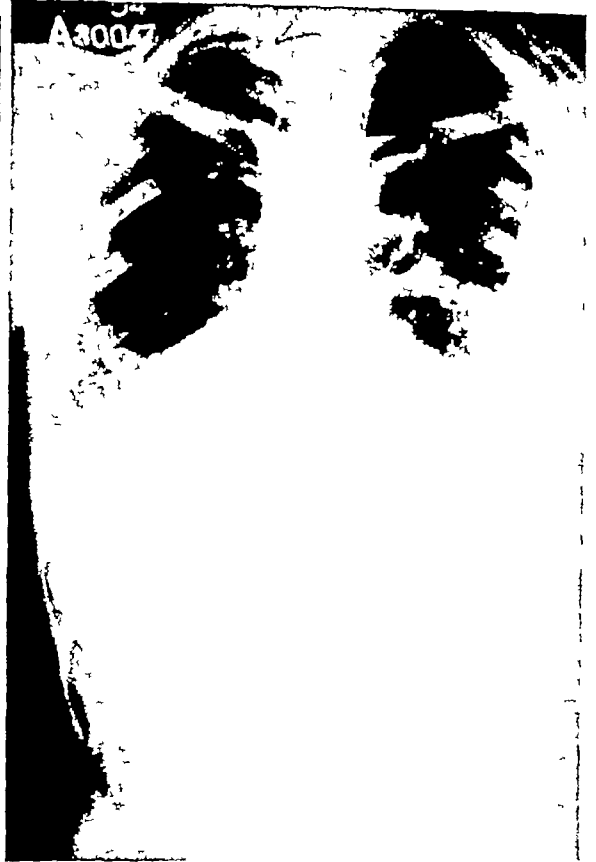


Fig 2B Same patient (Mar 2, 1954), after massive hemoptysis and bilateral spread of tuberculosis

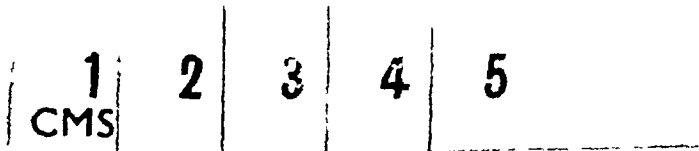


Fig 2C Specimen showing lesion after resection seven months later

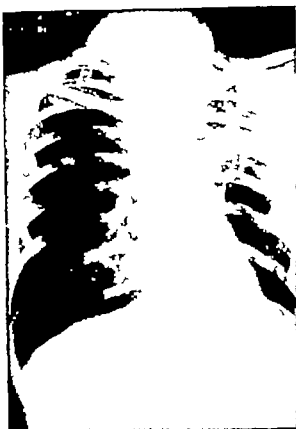


Fig. 3A. Left upper lobe bronchiectasis (Feb. 16, 1949) treated by resection and thoracoplasty



Fig. 3B. Same patient, Dec. 16 1955 massive hemoptysis from a now bronchiectatic left lower lobe emergency lobectomy

### CLINICAL FEATURES

Clinically the onset is usually sudden rarely with a small warning hemoptysis. The bleeding is either intermittent or continuous.

When first seen by the medical practitioner the patient is usually sitting up in bed—pale and anxious and coughing or spitting blood and saliva into a large bowl. Cyanosis is also present in those unable to clear their trachea and bronchi by coughing. Fever is later added from the pneumonitis that follows the scattered atelectasis. The pulse is of poor volume there is a low blood pressure and examination of the chest confirms the moist sounds conducted from retained endobronchial blood.

**Investigation.** There are three fundamental investigations:

- 1 Blood examination
- 2 Chest roentgenograms
- 3 Bronchoscopy

**BLOOD EXAMINATION.** In addition to blood grouping and hemoglobin estimations, it is advisable to assess the serum proteins and the albumin globulin ratio as a guide to the effects of the hemorrhage and disease on the patient's blood chemistry.

**CHEST ROENTGENOGRAMS.** These should be taken promptly. Posteroanterior views reveal any underlying lung lesion such as bronchiectasis, lung abscess, neoplasm or tuberculosis. The areas of aspiration atelectasis give widespread shadowing.

In the presence of intermittent hemoptysis it may be possible to do a bronchogram in a quiescent phase so as to outline the affected lobe or segments, and to check the integrity of the remainder of the lung.



Fig 3C Specimen removed at lobectomy.

**BRONCHOSCOPY** The *objects* of bronchoscopy are:

- 1 To find from which lobe of the lung the hemorrhage is occurring,
- 2 To block off the draining bronchus temporarily, arrest the bleeding, and allow of definitive surgical resection

Bronchoscopy is of greatest value during the stage of active bleeding, when, though the actual source of the bleeding may not be seen, the lobe of the lung from which it is rising is accurately determined. Unless this information can be obtained, the examination is worthless. Further, it allows removal of retained endobronchial blood clot.

*Technic.* As it is important to preserve a good cough reflex, bronchoscopy should be performed with the minimum of local anesthetic.

If the patient is at all dyspneic, he should be kept sitting up—at least until the obstructing sputum and blood clots have been removed. When he can tolerate it, on the other hand, he is best placed in the head-down position, thus allowing of bronchial

drainage. It is most important to have several bronchial suction tubes available during the procedure, lest one block and the patient drown in blood.

The bronchoscope is gently inserted, all blood and blood clot are removed by suction and large grasping peanut forceps and the affected bronchus viewed. When bleeding has ceased, little more may be found. When severe bleeding continues, however, or has been reactivated by the bronchoscopy, the aim is to control it temporarily and allow permanent measures to be taken.

If the bleeding is from either of the lower lobes or the middle lobe it can be controlled by inserting a Thompson cuffed endobronchial blocker. If this is not available, the orifices can be tightly packed with cotton swabs thereafter intubating the contralateral lung and proceeding to urgent thoracotomy.

If the bleeding is from either upper lobe—neither of which is at all easy to intubate effectively—it is safer to insert a cuffed McGill tube into the contralateral lung, in flate and seal off the hemorrhage.

Because of these possibilities bronchoscopy for massive hemoptysis is best performed in the operating room with everything ready for immediate thoracotomy.

### CLINICAL TYPES OF HEMOPTYSIS

Patients requiring treatment for hemoptysis fall into three clinical groups

1. Those with mild hemoptysis
2. Those with severe intermittent hemoptysis
3. Those with severe continuous hemoptysis

**Mild Hemoptysis.** This may vary from an occasional staining of sputum up to 10 ml. of blood appearing spasmodically. The object of the investigations which include chest roentgenography tomography bronchography and bronchoscopy is to find and treat the lesion, be it pulmonary cardiac, hypertensive or a manifestation of systemic disease. These details, however are beyond the scope of this book.

**Severe Intermittent Hemoptysis.** This is almost invariably caused by pulmonary tuberculosis, broncholithiasis, bronchiectasis lung abscess, or neoplasm. The condition of these patients usually allows sufficient time and opportunity for adequate assessment as a prelude to exploratory thoracotomy (2, 8, 9, 10, 11). Blood loss must be replaced. As much as 12 liters have been required (12). The lesion is then promptly removed by segmental, lobar or pulmonary resection.

**Severe Continuous Hemoptysis.** These patients form the most difficult group, for their situation constitutes a dire emergency and usually allows time for only a straight chest film to detect the causative lesion. Review of all former roentgenograms, if available is of great value.

There is no use in temporizing even for a day, for though the bleeding may cease momentarily it will return at a more inconvenient time. As lung function is invariably reduced from aspiration atelectasis leading to areas of localized pneumonia, conservative treatment is fraught with danger to the patient's life. Such preliminary conservative measures as pneumothorax, pneumoperitoneum or thoracoplasty are an unrealistic approach, as they merely reduce ventilatory function still further and reduce the chance of survival before, during, or after surgical operation. The decision to operate must be made early and, when made, carried out promptly.



## MANAGEMENT

Although a surgeon should be chary of advising operation in bilateral tuberculous cases, life is in danger, and the bleeding must take priority over conservatism. Ross (2) reported successful emergency pneumonectomy in the face of an acute exacerbation of tuberculosis.

**Preliminary Assessment.** The patient is examined, his chest roentgenograms scrutinized, and he is placed in the position for postural drainage of the affected segment in order to clear blood from the lungs. To preserve the cough reflex, only mild sedatives are given. The blood group and hemoglobin level are estimated and transfusion commenced.

**Bronchoscopy.** When bleeding continues unabated, bronchoscopy is performed in the operating room as a prelude to thoracotomy. Following the bronchoscopy and detection of the bleeding point, the sound lung is protected from aspiration of blood and clot by one of three ways.

- 1 By inserting a Thompson bronchus blocker into the affected lobe. This is of greatest value when bleeding comes from a lower lobe,
- 2 By packing the affected bronchial orifice with a cotton swab,
- 3 By intubating the sound lung with a cuffed McGill endobronchial tube. The anesthetist then takes over and induces general anesthesia as for thoracotomy.

Rarely, bleeding may be so severe that immediate bronchoscopy in the ward is required as a lifesaving measure. The torch-handle pattern bronchoscope (see Chapter 2, Fig. 12) is most adaptable and may have to be left in position as an airway while the patient is taken to the operating room and the hemorrhage arrested by bronchial blocking as described above.

**Anesthesia.** With the bleeding controlled and the danger of drowning abated, general anesthesia is now safely induced. The patient is turned on his side in preparation for thoracotomy, with his head low for postural drainage and tracheal toilet. In young children, in whom bronchus blocking is not possible, Overholt's (13) face-down position is required.

**Technic of Operation.** The chest wall is opened widely and any collapsed or destroyed lobe or calcified hilar lymph nodes recognized. It is at this stage that the full value of preoperative bronchoscopy becomes obvious, for, having seen blood issuing from the affected bronchus, the surgeon can approach the problem of lobar resection confidently and not be confused by the surrounding areas of aspiration atelectasis. Further, the bronchoscopy aids in deciding the extent of resection required.

The *extent of the resection* is based, therefore, on the preoperative bronchoscopic findings and on the presence at thoracotomy of an obviously destroyed lobe and/or calcified peribronchial lymph nodes.

The *operative procedure* depends on the findings.

1. When there is a destroyed lobe from bronchiectasis, lung abscess, or tuberculosis, the affected segments or lobes only are resected,
2. When calcified lymph nodes are eroding the bronchus, it may be possible to enucleate them. If not, then resection is necessary, the extent depending on the lesion. When the lymph nodes are firmly adherent to the main bronchus or pulmonary artery, for technical reasons pneumonectomy may be the only possible course, a finding Ehrenhaft (3) encountered twice in his series,

- 3 When thoracotomy fails to reveal a cause for the bleeding, the nature and extent of the resection depends entirely on the preoperative bronchoscopy and, at the least, resection of that lobe is required.

**Postoperative Treatment.** This follows the plan already outlined in Chapter 3

As Ehrenhaft stresses provided the preoperative localization of the site of bleeding has been accurate and this area has been resected, the postoperative convalescence should be uneventful. He reported no significant morbidity and no mortality in his group of 12 cases in whom the following operations were required

OPERATION	NUMBER OF CASES
Left pneumonectomy	2
Right upper lobectomy	2
Left upper lobectomy	2
Right lower lobectomy	2
Left lower lobectomy	1
Lingulectomy	1
Lingulectomy and basal segmental resection	1
Resection of calcified node, left main bronchus	1

### CONCLUSIONS

In such urgent resections success depends on

- 1 A clear-cut decision to operate
- 2 Roentgenography and bronchoscopy to localize the lesion
- 3 Adequate blood transfusion, especially while operating
- 4 Endobronchial tamponade to control the hazard of aspiration atelectasis in both lungs
- 5 Speed—these patients cannot reasonably be expected to withstand prolonged operative procedures (2)
- 6 Postoperative antibiotic therapy

Especially with tuberculous patients while in general it is unwise to attempt lung surgery unless the other lung is free from disease or has a minimal stable lesion, operation should never be so delayed that massive contralateral spread can occur or until the patient has been allowed to sink to such a poor state as to be beyond surgical aid.

### REFERENCES

- 1 Ryan, T. C. and Lineberry W. T. Pneumonectomy for pulmonary hemorrhage in tuberculosis, *Am. Rev. Tuberc.*, 61 426 1950
- 2 Ross, C. A. Emergency pulmonary resection for massive hemoptysis in tuberculosis, *J. Thoracic Surg.*, 26 435 1953
- 3 Ehrenhaft, J. L. and Taber R. E. Management of massive hemoptysis not due to pulmonary tuberculosis or neoplasm, *J. Thoracic Surg.* 30 275 1955
- 4 Charr R., and Savacool, J. W. Hemoptysis and pulmonary arterial rupture, *Am. J. M. Sc.*, 199 641 1940
- 5 Maier H. C. Transthoracic removal of calcified lymph node causing hemoptysis by bronchial erosion, *Am. Rev. Tuberc.*, 65 206, 1952.
- 6 Moersch H. J. Clinical significance of hemoptysis, *J.A.M.A.*, 148 1461 1952.
- 7 Pratt, L. W. Hemoptysis, *Ann. Otol., Rhin. & Laryng.*, 63 296 1954
- 8 Abbott, O. A. Clinical significance of pulmonary hemorrhage. Study of 1316 patients with chest disease, *Dis. of Chest*, 14 824 1948
- 9 Douglass, B. E., and Carr D. T. Prognosis in idiopathic hemoptysis, *J.A.M.A.*, 150 764 1952.

- 10 Parker, E F Hemoptysis, its significance and methods of study, Dis of Chest, 21 677, 1952
- 11 Souders, C R , and Smith, A T The clinical significance of hemoptysis, New England J Med , 247 790, 1952
- 12 Tobin, J L , and Johnson, C R A case of massive pulmonary hemorrhage secondary to bronchiectasis, Dis of Chest, 30 342, 1956
- 13 Overholt, R H , and Woods, F M Prone position in thoracic surgery, J Internat Coll Surgeons, 10 216, 1947

## 12

### MANAGEMENT OF POSTOPERATIVE COMPLICATIONS IN PNEUMONECTOMY, LOBECTOMY, AND THORACOPLASTY

The general rules for pre and postoperative management of thoracotomy apply also to the operations of pneumonectomy lobectomy and thoracoplasty (see Chapter 3) Their complications however require prompt correction and are logically considered as thoracic surgical emergencies

#### PNEUMONECTOMY

**Historical Note.** It was not until 1931 that Nissen (1)—then in Berlin—successfully removed an entire lung In 1932 Haight (2) of Ann Arbor repeated this success in 1933 Graham (3) at St. Louis performed pneumonectomy for neoplasm and, in 1934 Mason (4 5) at Newcastle upon Tyne and Edwards (6) and Roberts (7) in London each had similar successes The operation is most commonly performed for bronchial carcinoma but is also of value in the treatment of bronchiectasis tuberculosis suppurative pneumonia, bronchial adenoma and other rarer lesions.

#### ROUTINE PROBLEMS AFTER PNEUMONECTOMY

Successful postoperative management depends upon

- 1 Centralizing the mediastinum
- 2 Controlling pleural effusion
- 3 Preventing pleural infection.

If these three points are carefully watched, then complications will be minimal

**Centralizing the Mediastinum.** This is done as the chest wound is being closed, by having the anesthetist fully inflate the remaining lung After the patient is turned on to his back, intrapleural pressures are measured with an artificial pneumothorax needle inserted through the second intercostal space and adjusted to zero The final state is checked by a chest roentgenogram A water-seal drain is a useful safeguard against intrapleural pressure changes or a large pleural effusion In 100 pneumonectomies when this was standard practice no complication was directly attributable to the tube and it was lifesaving in several patients with severe postoperative hemorrhage. The water seal must be deep to prevent rapid expulsion of air from the pleural cavity via the tube, mediastinum shift toward the operated side, emphysema of the remaining lung respiratory distress and even cor pulmonale Should this happen, air is replaced with a pneumothorax apparatus and the mediastinum is centralized (Fig. 1A-C) The tube is removed when it becomes blocked, some 24 to 48 hours after operation, or earlier if indicated.



Fig 1A Immediate postpneumonectomy film with central trachea

**Controlling Pleural Effusion.** After pneumonectomy, fluid collects at a variable rate in the empty pleural cavity. For the first 10 days it is best kept below the level of the sixth thoracic vertebra by chest aspiration to allow optimal conditions for healing of the bronchial stump. The level is checked by regular roentgenograms (Fig 2). In some patients, especially children, little fluid collects, and no aspiration is required. In others, fluid accumulation may require daily aspiration. After the tenth to twelfth postoperative day, the fluid level is allowed to rise until it fills the hemithorax. In time, the accompanying fibrin becomes organized.

**Preventing Pleural Infection.** When pneumonectomy is required in the presence of a purulent pleural effusion (as sometimes occurs when carcinoma of the lung has caused a blocked-bronchus syndrome with empyema), the pleural cavity can be sterilized before operation by daily aspiration and instillation of the appropriate antibiotic.

Following operation on these potentially infected cases, the antibiotic regime is continued, 1 mega of penicillin, 1 gram of streptomycin, and so on (according to the



Fig. 1B Trachea displaced to left, following expulsion of air through intercostal tube.

sensitivity of the organism) being injected daily through the second intercostal space anteriorly for at least one week after the temperature is normal and all cultures of the aspirate negative for growth of organisms

Systemic antibiotics and adequate physiotherapy usually ensure freedom from infection in the remaining lung, which should be examined clinically each day

#### MANAGEMENT OF COMPLICATIONS OF PNEUMONECTOMY

In general complications are either *early* or *late*

*Early complications include*

- 1 Postoperative shock from hemorrhage
- 2 Tension pneumothorax
- 3 Surgical emphysema
- 4 Acute pulmonary edema
- 5 Sputum retention
- 6 Auricular fibrillation.



Fig 1C Trachea again centered, following introduction of air with pneumothorax needle

*Late complications are*

- 7 Fistula formation,
- 8 Pyothorax,
- 9 Disruption of the intercostal layer,
10. Wound infection (already described under Thoracotomy),
- 11 Pulmonary embolism,
- 12 Extrusion of a stitch from the bronchial stump

**Hemorrhage.** Postoperative hemorrhage may be either (a) sudden and profound, or (b) a slow steady ooze

The first type is rare, but it has occurred from slipping of ligatures on the divided pulmonary vessels. It is usually fatal. Treatment is prevention and is bound up in careful ligation of the main vessels and oversewing their stumps at operation.

The second type usually comes from divided parietal or mediastinal adhesions. Again, careful hemostasis before closing the pleural cavity is the surest means of prevention. For slow hemorrhage, repeated transfusions are necessary. If bleeding continues, the patient is taken back to the operating room, the wound reopened, the blood clot evacuated, and the bleeding point sealed.

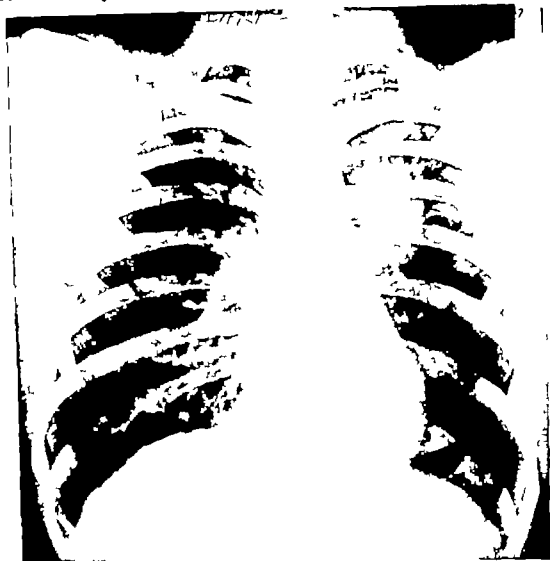


Fig 2A. Neoplasm of lung.

In one patient who drained 4 liters of blood in eight hours after pneumonectomy at emergency reopening of the wound a small bronchial artery was ligated. In another who bled after postoperative pleural aspiration and who failed to respond to 12 pints (8 liters) of blood given over 6 days the wound was reopened, and a steady parietal ooze was checked by pericostal ligation of the corresponding intercostal artery.

**Tension Pneumothorax.** When intrapleural pressures have not been checked and adjusted to zero at the end of the operation an existing tension pneumothorax may go undetected until the patient suddenly collapses. Deviation of the trachea gives the key to diagnosis. The intrapleural pressure may also rise later from intrapleural hemorrhage or from rapidly increasing pleural effusion. Tension pneumothorax from either cause results in shift of the mediastinum toward the remaining lung, decreased pulmonary ventilation, acute respiratory embarrassment, and hypoxia. If the pneumothorax is due to hemorrhage, there are also the signs and effects of blood loss. Rapid improvement follows centralizing of the mediastinum and replacement of the blood loss.

**Surgical Emphysema.** This occurs following failure to approximate the intercostal muscles completely when pieces of several adjoining ribs have been resected, or after using an intercostal muscle flap to cover the bronchial stump.





Fig 2B Ten days after pneumonectomy, left pleural effusion to level of sixth rib

cough, air is expelled from the pleural cavity into the muscular and subcutaneous planes of the chest wall. Though alarming, surgical emphysema is usually not serious and is best controlled by firmly applying a cotton pad with elastoplast to the intercostal gap. Rarely, the emphysema may be severe and cover the entire body, as in Figure 6 of Chapter 4. It can be relieved, and the air allowed to escape, by applying an antiseptic solution and making multiple skin punctures with a sharp-pointed scalpel blade.

**Acute Pulmonary Edema.** The exact cause of this condition is unknown. It usually occurs a few hours after operation in elderly patients with some degree of emphysema and persistent pulmonary hypertension when circulatory adjustment after ligating the pulmonary artery was delayed. A "mobile" mediastinum may aggravate the condition. Occasionally, the pulmonary edema may be delayed in onset until the end of the second week. It is treated by morphine, intravenous atropine 0.65 mg six-hourly, mersalyl, oxygen, and intratracheal suction, preferably by bronchoscopy. If this procedure requires repeating, tracheotomy should be done.

**Sputum Retention.** Not infrequently within 12 hours of operation, especially in the elderly, the patient is unable to clear his trachea of sputum. He becomes pale and sweating and his blood pressure falls. If unrelieved, his condition will deteriorate.

rate further become irreversible, and prove fatal. *Experience has shown it is useless to wait for x-ray evidence of atelectasis before taking action.* Bronchoscopy is urgently required when this must be repeated tracheotomy is indicated. Recent experience has proved the special worth of early tracheotomy in the postoperative management of pneumonectomy.

**Auricular Fibrillation.** In elderly patients, too vigorous ambulation not infrequently precipitates auricular fibrillation. A recent group of 3 patients, all over 70 years of age, developed auricular fibrillation on the third postoperative day. It may occur whether the pericardium has been opened or not, and after esophageal or cardiac operations as well as pleural lesions. The fibrillation is usually transient and responds rapidly to bed rest and quinidine in doses of 200 mg. 3 hourly for 3 doses then 400 mg. 3 hourly for 3 doses and so on, until the rhythm is again regular (23).

**Postoperative Bronchial Fistula.** This most serious complication of pneumonectomy is largely preventable by

- 1 Meticulous closure and covering of the bronchial stump with a viable pleural flap or intercostal muscle bundle and carefully burying it in mediastinal tissue
- 2 Careful postoperative chest aspiration, ensuring that the level of intrapleural fluid is kept below the bronchial stump until healing is complete

Bronchial fistula may occur early or late in the postoperative period. Postoperative bronchial fistulas usually arise during the second week but have been known to occur as long as three months after operation. They may be either small or massive.

**Small Fistula.** In some patients, the presence of a small fistula may be difficult to prove. Some guide is possible by measuring intrapleural pressure, aspirating 500 cc of air, taking pressures again, and rechecking 10 minutes later. If the pressure has by then returned to the original level a fistula is present. The presence of a fistula may also be demonstrated by injecting 5 ml. of methylene blue solution into the pleural cavity and noting its appearance in the sputum.

Because of the ever present danger that small fistulas can become massive and because of the associated intrapleural infection, there is nothing to be gained by tamponading. The pleural cavity requires prompt draining preferably by rib resection evacuation of all fluid and fibrin clot and closure with water seal drainage. Thereafter thoracoplasty is required.

**Massive Fistula.** The patient, who for some days may have had a smouldering temperature or occasional bloodstaining of his sputum, is seized with a violent fit of coughing and expectorates the greater part of the pleural fluid. The danger here is death from drowning in the fluid suddenly released into the bronchial tree.

The patient should immediately be placed in the head-down position and lying on the operated side. If at home, he should be returned at once to hospital lying in that position en route. As soon as possible an intercostal catheter with water-seal drainage is inserted into that pleural cavity and the patient is bronchoscoped. The fistula may in time close but the now infected pleural cavity will not heal unless completely obliterated by total thoracoplasty. In the young, such an operation is well tolerated and compatible with a useful working life. In those over 50 however although the operation is well tolerated the patient seldom can return to a previous heavy occupation. He is, however fit for most sedentary work.

**Pyothorax.** Pyothorax arises from

- 1 Infection present before operation,
- 2 Infection introduced during operation,
- 3 Postoperative wound infection,
- 4 Bronchopleural fistula

Because of previous antibiotic therapy, diagnosis may be delayed and difficult. Rapid reaccumulation of pleural fluid may suggest the correct diagnosis, while culturing the organisms from the aspirate proves it.

**Conservative Management** When there is no bronchial fistula, every attempt should be made to control the infection by repeated aspiration and injection of the appropriate antibiotics. When the fluid is loculated, each loculus requires aspiration. Attempts can be made to liquify the fibrin with streptokinase-streptodornase. Faced with an organism, usually a staphylococcus insensitive to both penicillin and streptomycin, Griffin (8) in 1951 had satisfactory results by reopening the chest, manually evacuating the fibrin clot, cleansing the pleural cavity with eusol and saline, and finally leaving antibiotics in the cavity before closing. The appropriate antibiotic was continued systemically and intrapleurally, and the pleural space was regularly aspirated until the temperature had been normal for one week and all cultures of the aspirate rendered sterile.

**Drainage** If these conservative measures fail, then rib resection is performed and a water-seal drain inserted well forward in the anterior axillary line. When the patient's condition has improved, the space is collapsed by thoracoplasty.

**Disruption of the Intercostal Layer.** Occasionally, when continuous sutures have been used, the intercostal layer gives way. With each cough, pleural fluid enters the muscle layers of the chest wall and the wound bulges. Unless it is treated, the wound may rupture. A large pad strapped firmly to the chest wall for a month will ensure support until the periosteum reossifies and rigidity returns.

**Wound Infection.** See Thoracotomy, page 52.

**Pulmonary Embolism.** This is usually fatal. In a study of 200 lung resections for carcinoma of the lung, the author found that pulmonary embolism accounted for 21 per cent of postoperative deaths. All of the emboli came from the leg veins (9).

In five or six patients, pulmonary embolism occurred without warning: one on the third, two on the ninth, and one each on the twelfth and thirty-ninth postoperative days, respectively. After a warning femoral thrombosis, the sixth patient received anticoagulant therapy, but this failed to prevent the fatal embolus, which occurred on the sixty-first postoperative day.

When calf-vein thrombosis is present, anticoagulant therapy and oxygen are immediately indicated. With sudden syncope, if a doctor is available, transthoracic cardiac compression is indicated, since in an appreciable proportion of cases the embolus is small and has not completely blocked the pulmonary artery but has precipitated ventricular fibrillation (see Chapter 24).

When the clot is massive, Trendelenburg operation will only rarely be successful, for the lethal part of the embolus is the most distal part and the most difficult to grasp.

**Extrusion of a Stitch from the Bronchial Stump.** Sometimes, after pulmonary resection the patient returns with a history of brisk hemoptysis or dry, irritating cough. Check bronchoscopy may show an extruded stitch lying in the bronchial stump. It is removed with grasping forceps.

## LOBECTOMY

**Historical Note.** One of the earliest lobectomies was performed in 1913 by Morrison Davis (10) His patient lived for 10 days after operation

In treating bronchiectasis, the technic of tourniquet lobectomy (5 11 12) was replaced in 1939 by dissection lobectomy evolved by Churchill and Belsey (13) Since then, with the wider use of chemotherapy and postural drainage based on an appreciation of bronchial anatomy consistently good results have been obtained.

Lobectomy for pulmonary tuberculosis however was fraught with added problems which were not finally overcome until the development of endobronchial blockers such as the Thompson blocker The face-down position for the patient on the operating table (14 15) prevented spilling of sputum during operation and the free availability of streptomycin since 1948 has lessened spread of the disease in the post operative period

Lobectomy may also be required for chronic lung abscess adenoma, and for some patients with bronchial carcinoma.

**Postoperative Treatment: Object and Problems.** The aim of postoperative treatment and the chief postoperative problem following lobectomy is to regain and maintain full expansion and function of the remaining lung tissue a task that may tax the resources of an entire thoracic surgical team The vicissitudes of residual lobes and the detail of keeping them aerated can be adequately learned only by working in a thoracic surgical unit and observing the patients night and day The *problems* include

- 1 Management of the pneumothorax and closure of the chest wall,
- 2 Maintaining aeration of the remaining lobe
- 3 Management of complications

MANAGEMENT OF THE PNEUMOTHORAX AND CLOSURE  
OF THE CHEST WALL

When the lung resection has been completed and the remaining lobes re-expanded by positive pressure there are almost invariably present small residual parenchymal fistulas These require intercostal drainage to remove the pneumothorax, for without such a device the remaining lobe could never be made to function. Usually, two tubes are inserted, the site varying with the surgeon's preference In general, one tube acting as a drain lies in the posterior axillary line while the other tube acting as an air vent is led to the apex of the pleural cavity Both are connected with water-seal drainage bottles and continuous suction. If the air leak proves large suction should be applied in the operating room. When the patient is moved from the operating table to his bed or back to the ward these two drains must be kept dependent and freely functioning, for raising them above the level of the patient allows fluid to be aspirated into the pleural cavity If the tube is raised high, it must be clamped.

**Bronchoscopy** At the end of the operation, after careful nasal and pharyngeal aspiration of mucus the endotracheal tube and any bronchus blocker are removed. A bronchoscope is then passed and any secretions completely aspirated.

**Immediate Roentgenogram.** Immediately following bronchoscopy, a chest roentgenogram is taken with a portable x-ray apparatus to establish at the outset a baseline of lobar expansion. Thereafter, regular films are taken until it is certain that all remaining lung tissue is well expanded.

### MAINTAINING AERATION OF THE REMAINING LOBE

This can prove as great a task as the operation itself; it is closely related to

- 1 Patent bronchi kept clear by effective postural cough and expectoration of sputum;
- 2 Relief of pain by sedation and intercostal local anesthesia;
- 3 Use of patent intercostal tubes to allow escape of both fluid and air from the pleural cavity,
- 4 Continuous suction

**Eradication of Nasal Sepsis and Reduction of Sputum.** In treating bronchiectasis, the principle is usually accepted that no lobectomy should be performed until every attempt has first been made to eradicate nasal sepsis and to reduce sputum as much as possible—certainly to less than 1 oz. per day—by vigorous preoperative physiotherapy and chemotherapy

Again, it is most unwise to perform lobectomy unless the patient can cooperate well, particularly by postoperative coughing. Although children between the ages of five and nine years have had successful lobectomies for bronchiectasis, they do tend to have a more troublesome convalescence than older patients. Therefore, whenever possible, operation should be delayed until the older age-bracket is reached.

Twice each day after lobectomy, patients should have postural drainage and a period of coughing with the affected side of the chest firmly supported by a physiotherapist. Throughout the day, too, they must be encouraged hourly by the nursing and medical staff to cough and to breathe deeply. Sedation increases cooperation.

**Intercostal Drainage.** From the time intercostal drains are inserted, they require careful, regular attention. Any sudden jerk when the patient is being lifted onto his bed may pull a drain into, or even out of, the chest wall. During recovery from anesthesia, the patient should lie so as to allow free pleural drainage and uninterrupted “swing” of the fluid with each breath. He is thereby also in the correct position for postural drainage. When the patient has recovered consciousness and has been propped up in bed, all concerned in his management must see that he does not lean on the drain and block it. It should be “milked” down at least once every hour to avoid clot retention and blocking. Even when a lobe of lung is collapsed, a tube may still “swing” with each inspiration. However, the “swing” is then never smooth and easy but suspiciously jerky.

The drainage is at first heavily blood stained and amounts to 15 to 20 oz. in the first 24 hours. Thereafter, the lower drain usually blocks with fibrin and is removed.

It is essential to have daily roentgenographic checks, as well as clinical evidence of maintained expansion of the lobe.

**Continuous Suction.** When applied to the intercostal drain and run at a pressure of 20 mm. of mercury, continuous suction creates a constant negative intrapleural pressure and removes all air escaping from parenchymal fistulas. One method is illustrated in Chapter 2, Figure 6. The suction should be attached to the lower tube first, and, when this blocks, moved to the upper one.

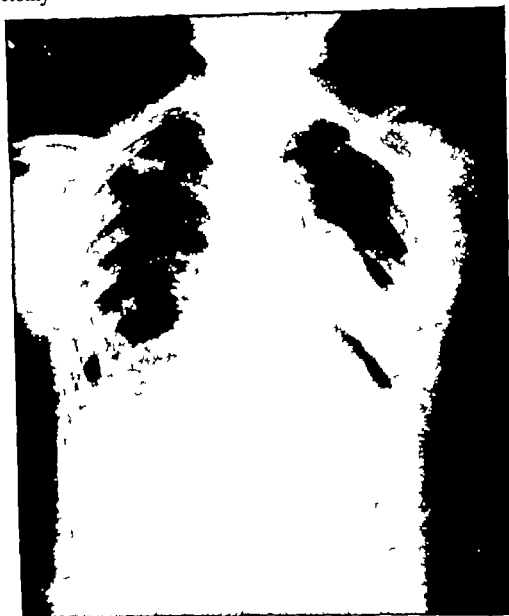


Fig. 3A. Following lower lobectomy for bronchiectasis, upper lobe is atelectatic because of pneumothorax developing after blocking of intercostal tube.

When the parenchymal fistulas close and therefore a swinging tube ceases to bubble off air or when the tube ceases to swing and the roentgenogram shows a fully expanded lobe the remaining intercostal drain is removed. Subsequent pleural effusion is aspirated as required.

Physiotherapy is continued as long as the patient remains in hospital.

#### MANAGEMENT OF COMPLICATIONS

The complications of lobectomy include

- 1 Collapse of the remaining lobe and persistence of parenchymal fistulas
- 2 Late bronchial fistula
- 3 Persistent collapse and destruction of the remaining lobe
- 4 Pleural effusion and hemothorax
- 5 Pleural infection.

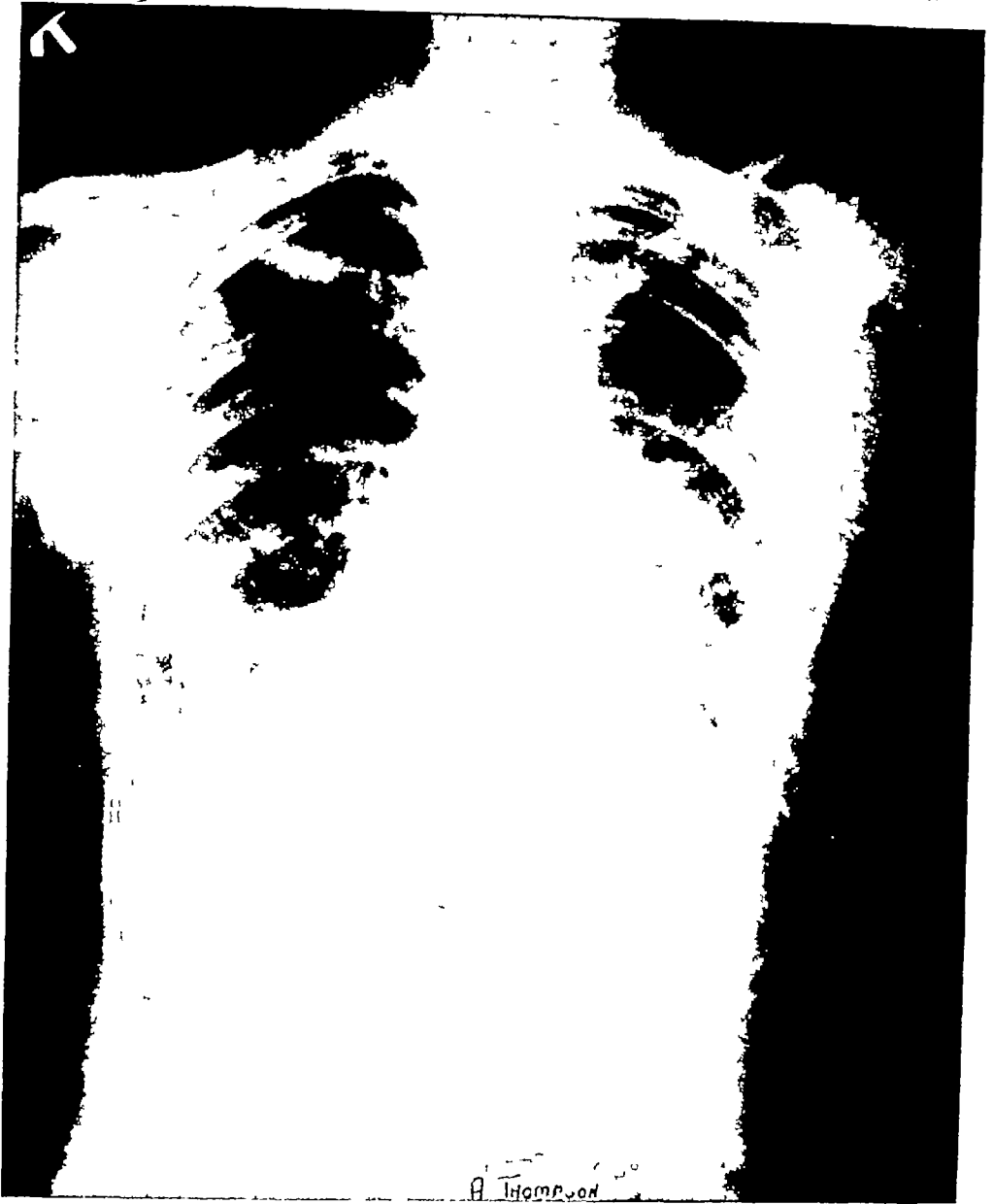


Fig 3B Lobar re-expansion after changing tube

**Collapse of the Remaining Lobe.** Following lobectomy, the most common complication is collapse of the residual lobe. There are several causes, including.

- 1 Blocking of the drainage tube with blood or serum,
- 2 Inability to cough up sputum because of pain or fear of pain,
- 3 Presence of persistent parenchymal fistulas,
- 4 Development of postoperative bronchial fistula

The clinical picture reveals an anxious patient, a moist cough, a poorly moving chest wall, and no air entry. Chest roentgenograms confirm the clinical diagnosis.

Collapse of the lobe can largely be prevented by regular postoperative coughing, starting as soon as the patient is conscious, careful removal of tubes and bottles, and adequate aspiration of all postoperative intrapleural fluid and air.

It is important to know the "overnight" state of the lobe early each morning, for if the lobe is collapsed, a series of steps lasting over three to four hours may have to be taken before re-expansion occurs. The cooperation of the x-ray staff taking "portable" films is, therefore, of utmost importance.



Fig. 4A. Left lower-lobe bronchiectasis.

**BLOCKED TUBE** When the tube has blocked and the roentgenogram shows fluid and air in the pleural cavity the intercostal drain is milked, if that fails to clear it, then a fresh intercostal drain is inserted (see Chapter 2) Combined with vigorous physiotherapy this change of tube may produce re-expansion of the lobe (Figs 3A and B)

**BLOCKED BRONCHUS.** When because of pain, the patient will not cough, and when roentgenograms show a collapsed lobe the intercostal drain is first checked for patency Thereafter if postural drainage fails to dislodge the retained sputum aspiration bronchoscopy is performed and the result checked by x-ray (Fig 4A D) Even



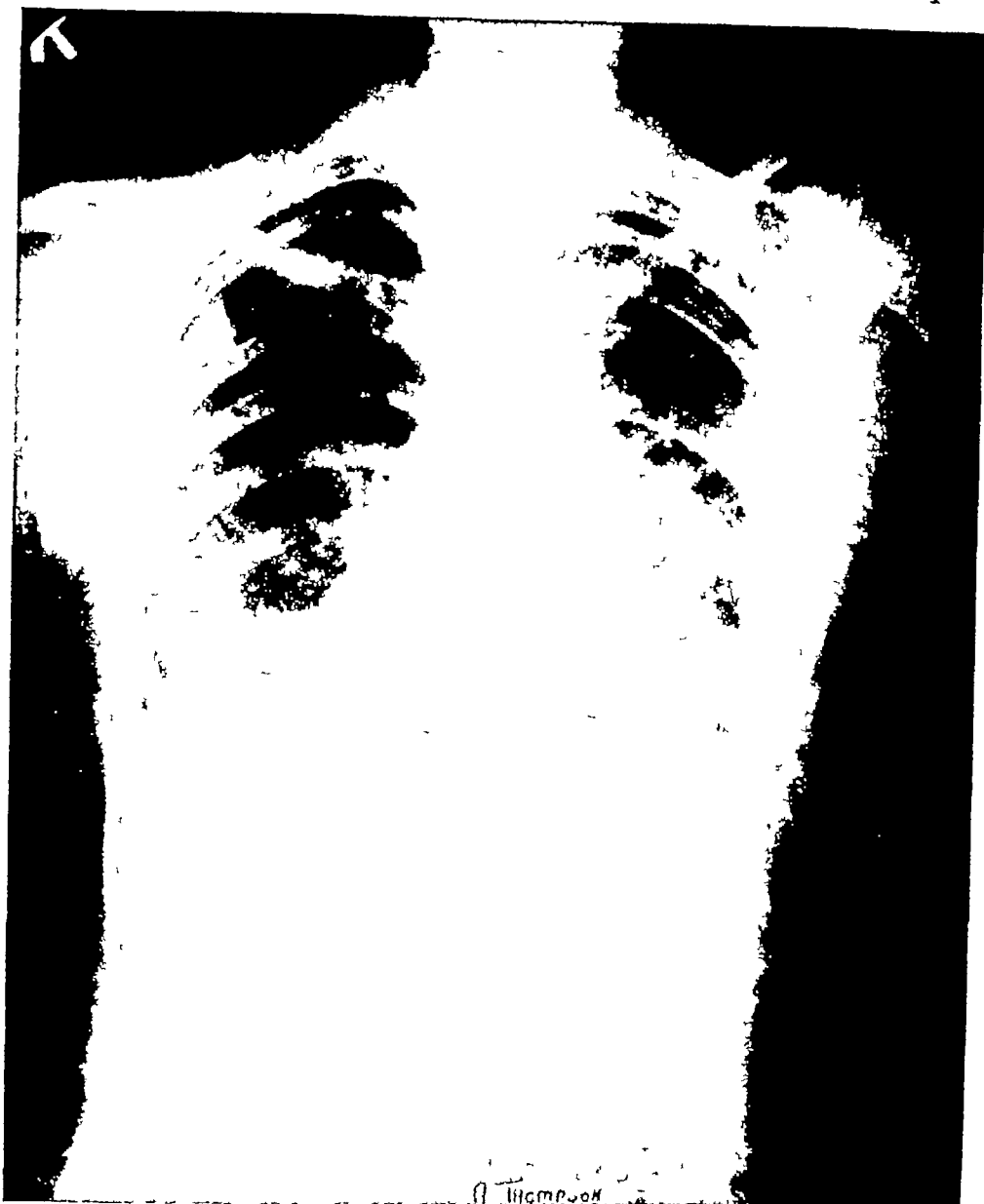


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Fig. 4A. Left lower lobe bronchiectasis.

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**BLOCKED BRONCHUS** When, because of pain, the patient will not cough and when roentgenograms show a collapsed lobe the intercostal tube must check for patency Thereafter if postural drainage fails to dislodge the mucus, bronchoscopy is performed and the result checked by roentgenogram (Fig 4A-D). If



**Fig 4B** Immediate postoperative roentgenogram, same patient



Fig. 4C. Atelectasis from sputum-retention and pneumothorax



Fig 4D Re-expansion following aspiration bronchoscopy and pleural aspiration

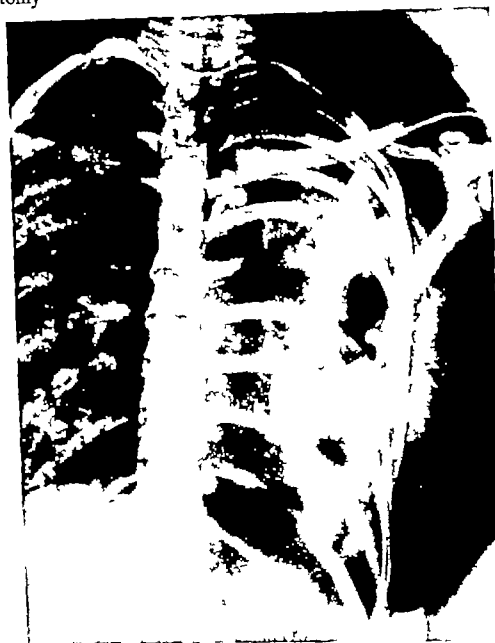


Fig 5A. Atelectasis of upper left lobe after left lower lobectomy (Note the outline of the trachea, left main bronchus and intercostal tube)

after bronchoscopy further vigorous postural drainage and coughing may be required to get the lobe fully re-expanded (Figs. 5A and B)

Two of the most stubborn causes of collapse of the remaining lobe after lobectomy are

- 1 Persistence of parenchymal fistulas
- 2 Development of bronchial fistula at the line of bronchial division (see Late Bronchial Fistula, below)

*Persistence of Parenchymal Fistulas* Provided there is free communication between a parenchymal fistula and an intercostal drain, the lobe will remain expanded. In some lobes however when the intercostal drain has become blocked or has been removed too early there is at first apparent full lobar expansion followed by an atelectasis that resists all efforts at re-expansion.

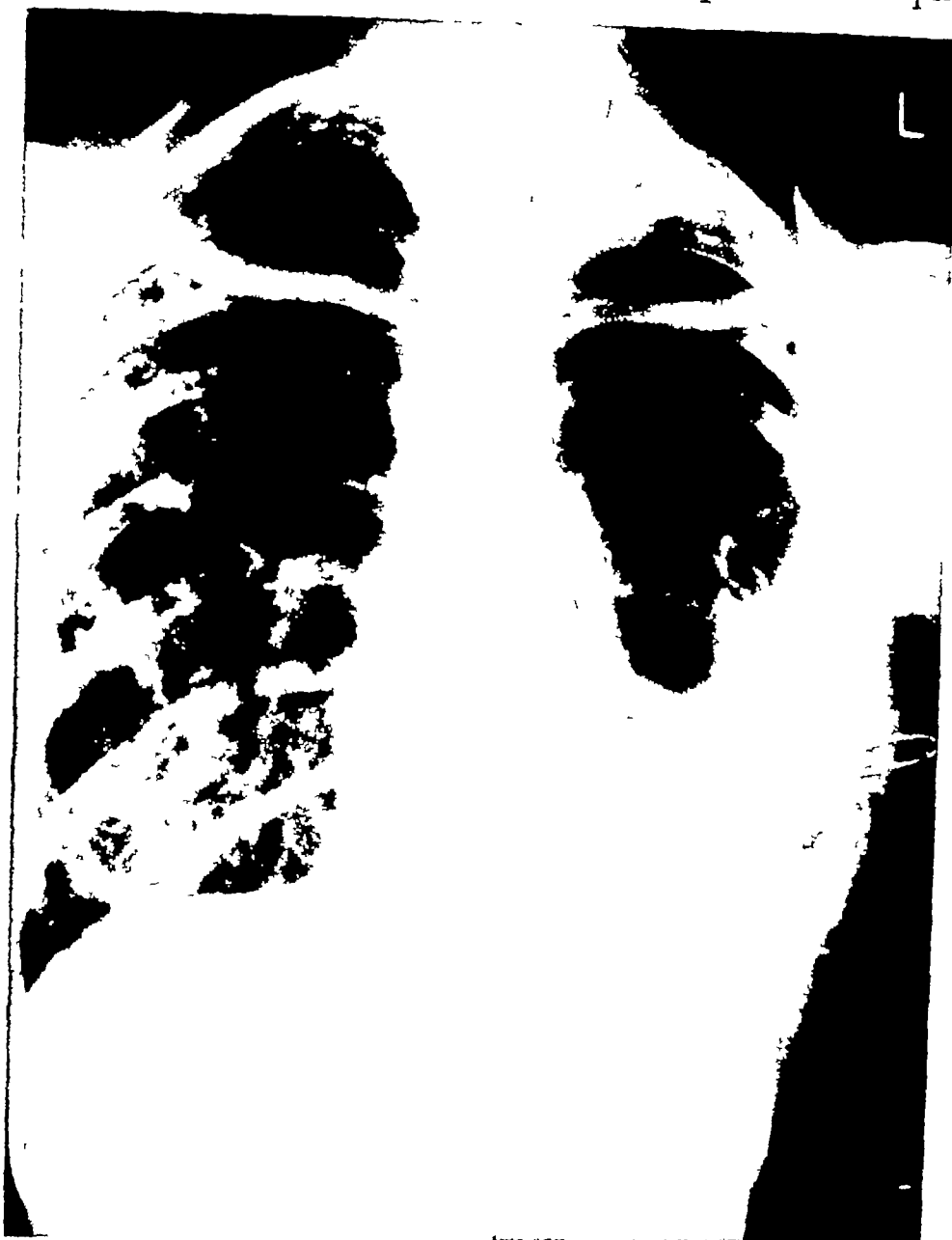


Fig 5B Upper lobar re-expansion following effective postural drainage by physiotherapists

Roentgenograms do not readily show the optimum site for replacing intercostal drains. In these resistant cases, recent—if rare—experience has shown that reopening the wound, re-expanding the lung, using endotracheal anesthesia, and redrainage constitute the treatment of choice. The lobe is re-expanded, and the fistulas are properly drained, thereby avoiding more serious complications.

**Late Bronchial Fistula.** This complication usually declares itself as follows:

The parenchymal fistulas may have closed, the lobe may have been clinically aerating, and the intercostal drain may have been removed, but the temperature may not have completely settled. Sometimes, during the second postoperative week the patient will cough up blood-stained sputum, and roentgenograms may demonstrate increased pleural fluid with a fluid level or lobar collapse. The usual cause is a fistula developing at the site of bronchial division. If the fistula is basal, a loculated air pocket may be seen on a penetrating film. When a fistula is detected, this space should be

drained either by an intercostal tube or by rib resection with evacuation of any fibrin and clot and with subsequent water-seal drainage. In two weeks when pleural adhesions form the water seal is removed and a short tube inserted. The healing of the residual sinus is thereafter controlled with weekly sinograms to guide the shortening of the drainage tube.

**Persistent Collapse of the Remaining Lobe.** Rarely in non-coughing patients or in those patients with persistent parenchymal or late bronchial fistulas not recognized as such and not adequately treated, the collapsed lobe becomes infected by a severe type of "pneumonitis" that, in the short space of six weeks can transform the remaining normal lobe into destroyed bronchiectatic tissue. This is a serious problem usually seen only in the surgical treatment of bronchiectasis. The chief aim of postoperative care is to prevent this unwanted complication. Continued intrapleural drainage and tracheotomy to aspirate retained sputum are the keys to successful treatment.

When the lobe is destroyed it is best removed, which means a pneumonectomy must, therefore be performed although lobectomy was the original intention. This distressing complication is fortunately rare, but no one who has done a large series of resections for bronchiectasis in children has escaped it.

**Pleural Effusion and Hemothorax.** *Pleural Effusion* With efficient pleural drainage, suction, and rapid re-expansion of the remaining lung tissue pleural effusion is minimal. However when pneumothorax persists an effusion rapidly forms which can readily become infected. Fibrin deposited on the underlying lung further lessens the chance of easy re-expansion. Treatment is by early and complete aspiration.

*Hemothorax* Rarely following lobectomy, massive postoperative bleeding from an intercostal vessel from parietal adhesion, or from lung parenchyma may clot in the pleural cavity interfere with full lung re-expansion and later become infected. Hemorrhage may also follow chest aspiration. Although aspiration after instilling streptokinase-streptodornase has been successful, the only truly effective method is to reopen the wound, evacuate the hematoma, and close the wound again over water-seal drainage.

**Pleural Infection.** Because of the use of dissection technic for lobectomy as well as careful asepsis and antibiotics postlobectomy pleural infection—common a decade ago—is now rare. The space occupied by the resected lobe is rapidly obliterated, partly by the diaphragm and partly by mediastinal shift. Rarely however pleural infection does supervene on pleural effusion, usually secondary to bronchial fistula. The infection is treated at first by aspiration and local antibiotics but when that method is unsuccessful, treatment is by intercostal drainage or rib-resection drainage as for empyema.

When, after drainage of an infected space a bronchopleural fistula persists even after prolonged intercostal drainage it may not heal. In these cases, much time and inconvenience is saved by performing the "tram-line" operation described by Mason (22). The sinus is excised together with the ribs and parietal pleura over the empyema cavity which is thus saucerized. The intercostal muscle bundles and their blood supply are preserved, placed over the fistula, and firmly held there by packing and dressings. After 10 days, when the packing is removed the fistula will be found covered by a mass of granulation tissue which slowly heals.





Fig 5A Unilateral fibrocaceous disease with atelectasis of left upper lobe and tracheal displacement

### THORACOPLASTY

**Historical Note.** In the gradual evolution of the treatment of pulmonary tuberculosis, thoracoplasty was devised by Sauerbruch (17) for extensive unilateral fibrocaceous disease when the accompanying atelectasis had caused gross falling in of the ribs and mediastinal displacement (Fig. 6) In 1935, Holst, Semb, and Frimann-Dahl (18) described selective apical thoracoplasty with apicolysis for closing apical lung cavities. The technic involved freeing the scapula from behind by a periscapular



Fig. 6B. Same patient, after left Sauerbruch thoracoplasty—nine ribs.

incision and subperiosteal resection of all of the first and second ribs and of half of the third rib and performing apicolysis to the level of the first or second intercostal space in front and the fifth or sixth rib behind. At a second stage collapse of the cavitated upper lobe was enhanced by resecting lesser lengths of the fourth to seventh ribs. Further modifications giving a more stable chest wall, especially for use with lobectomy have been described by Borrie (19) (Fig 7) Björk (20) and Brock (21). A total thoracoplasty is occasionally required to collapse a postpneumonec-tomy empyema cavity.



7A Modified Holst thoracoplasty, preserving ribs two to five and using them to give stability to chest wall

### COMPLICATIONS OF THORACOPLASTY

Although the complications of thoracoplasty arise during the operation, their effects linger on into the postoperative period. The chief complications are.

- 1 Punctured pleura,
- 2 Hemorrhage,
- 3 Spontaneous pneumothorax in the contralateral lung,
4. Rupture of a tuberculous cavity into the extrapleural space

**Punctured Pleura.** This can arise with apicolysis when there is a thin fascial layer or a few apical pleural adhesions. It is more prone to occur with the coughing that may accompany the use of local anesthesia. The usual sites are near the second costochondral junction in front or the sixth rib behind. It is best avoided by utmost gentleness.

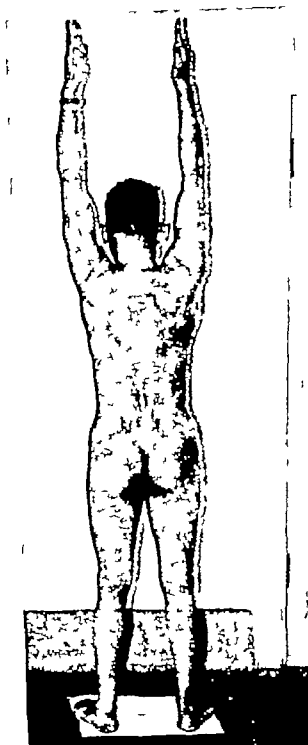


Fig. 7B Postoperative view of patient, showing full range of arm movement and no deformity

*If the puncture is small* it should be closed by clamp and suture. When the pleura proves friable and difficult to suture it is covered with a swab. The operation completed, the residual pneumothorax aspirated, and lung re-expansion checked by roentgenograms. Any subsequent pleural effusion requires aspiration.

*If the puncture is large* the safest and simplest treatment is to insert an intercostal catheter into the pleural cavity (e.g. in the eighth intercostal space in the posterior axillary line) and connect a water-seal drain and suction. Not only is the air removed but also the blood stained fluid seeping down from the extrapleural space. The catheter is removed when it ceases to drain, usually after two days.

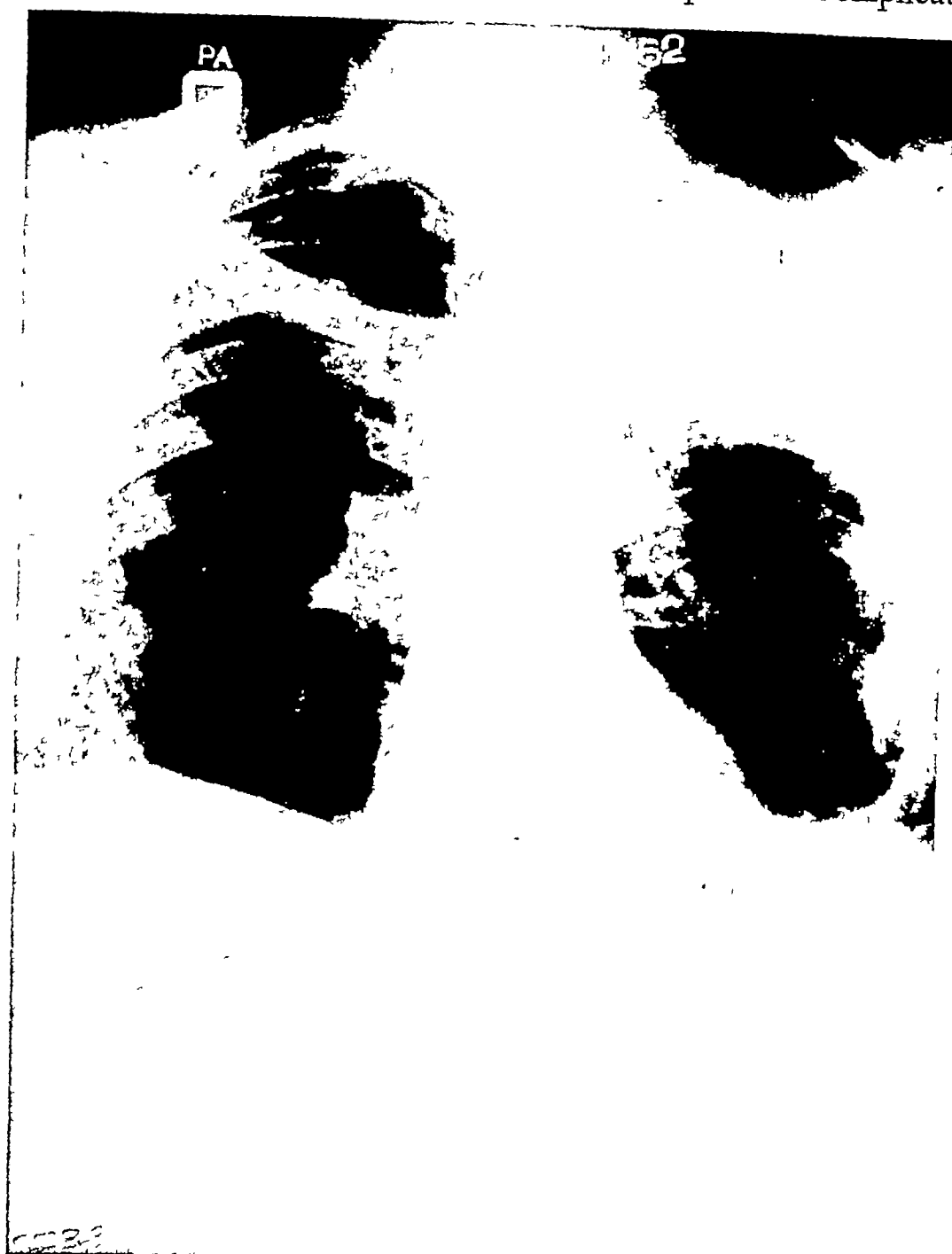


Fig 8A Hematoma developing after second-stage thoracoplasty

**Hemorrhage.** In patients with dense fibrous vascular adhesions holding the apex to the parietes, unless all bleeding points are carefully sealed before closing the wound, continued steady hemorrhage may complicate the early postoperative period. As such points cannot be readily ligatured, they are best closed by applying diathermy to the metal suction tip as it sucks the bleeding point. Finally, it may be necessary to cover the area with gelatin sponge. If an extrapleural hematoma forms after operation, it will require aspiration, and if large, evacuation (Figs 8A-C)



Fig 8B Hematoma showing left side opaque.



Fig 8C Radiotransparency restored after evacuation of hematoma

**Spontaneous Pneumothorax in Opposite Lung** This rare complication may occur in a middle-aged or elderly patient with a contralateral artificial pneumothorax and emphysema. The following case report illustrates the problem and its management.

**CASE REPORT** Mrs. S. aged 45 had bilateral pulmonary tuberculosis with an extensive right apical cavity and a lesion in the apical segment of the left lower lobe controlled by artificial pneumothorax (Fig 9A). In May 1952 one-stage right thoracoplasty was performed under general anesthesia. Toward the end of the apicectomy, the right pleura was punctured and air entered the underlying right pleural cavity. Because the anesthetist found difficulty in inflating the lungs, a right intercostal tube was inserted and connected to a water seal. The patient's condition did not improve after immediate postoperative bronchoscopy nor after the aspiration of 3 000 cc. of air from a tympanic left pleural cavity. A portable roentgeno-



Fig. 9A. Bilateral pulmonary tuberculosis, with an existing left pneumothorax.





Fig 9B Following right thoracoplasty and accidental opening of the right pleural cavity, patient developed left spontaneous pneumothorax

gram was taken. As satisfactory oxygenation could be maintained only by inflation with an anesthetic machine, and as the left chest was still tympanitic, a further 2,000 cc of air were removed without appreciable change. It was then realized that she had developed a left spontaneous tension pneumothorax during the operation. This was confirmed by the roentgenogram (Fig 9B) and by the rush of air and immediate relief that followed insertion of a Malecot catheter with water-seal drainage into the left pleural cavity. The left lung was finally re-expanded by continuous negative-pressure pleural suction (Fig 9C).



Fig. 9C. Left spontaneous pneumothorax relieved by intercostal water seal drain; right basal effusion required pleural aspiration.

**Rupture of Tuberculous Cavity into Extrapleural Space.** This equally rare complication may occur when the cavity to be treated is firmly adherent to ribs or spine.

*If the complication is recognized during the operation* the safest plan is to perform a resection, for suture of the tuberculous wall is hazardous and subsequent tuberculous infection of the extrapleural space almost inevitable.

*If the complication is recognized after operation* there is persistence of air in the extrapleural space and absence of fluid which will have drained away through the fistula thus created. The presence of a fistula is confirmed by injecting 5 ml. of methylene blue into the space and noting if it is coughed up. The simplest and safest treatment is a "letter-box" drainage of the extrapleural space between the axillary folds.

## POSTOPERATIVE COMPLICATIONS

Complications following operation are·

- 1 Postoperative shock,
- 2 Atelectasis of the lower lobe,
- 3 Excessive effusion into the extrapleural space,
- 4 Rupture of wound,
- 5 Wound infection and extrapleural-space infection,
- 6 Rupture of tuberculous cavity into extrapleural space,
- 7 Spread of disease (Not discussed )

**Postoperative Shock.** This occurs within 24 hours of operation from (a) failure to replace blood loss, and/or (b) failure to prevent paradoxical movement by adequate support of the chest wall

Blood loss after operation is detected by pallor, sweating face, dyspnea, rapid pulse small in volume, and falling blood pressure. It is usually due to continued oozing into the extrapleural space, treatment is by immediate and adequate blood transfusion

At the same time, the "paradox pad" is inspected, and, if its adhesive-plaster support has in any way slackened, it is firmly reapplied, the patient kept warm, and sedatives such as omnopon 22 mg given (Fig 10)

**Atelectasis of the Lower Lobe.** It is just as important to preserve a functioning lower lobe after an apical thoracoplasty as it is to preserve functioning lung tissue after lobectomy. For that reason phrenic nerve operations are not encouraged as a prelude to thoracoplasty. Postoperative deep breathing and coughing exercises are essential. If clinical examination and roentgenograms show mediastinal displacement and continued atelectasis of the lower lobe, aspiration bronchoscopy is required. Even after bronchoscopy, further physiotherapy may be required to re-expand the affected lobe completely.

**Excessive Effusion into the Extrapleural Space.** Occasionally a routine postoperative roentgenogram shows a blacked out lower lobe, although clinically good breath sounds may be heard. If a penetrating film is taken, it will confirm that air is entering that lobe. The shadowing is due to fluid from the extrapleural space seeping down into the extrapleural layer and simulating the x-ray appearance of atelectasis—usually, however, without tracheal displacement.

At other times, the extrapleural space may become tensely ballooned with fluid and air, and the patient experiences a sense of tightness in the area of the operation. The remedy is to aspirate the fluid at the most favorable place for the patient:

- 1 Through the axilla,
- 2 From in front, just below the midpoint of the clavicle,
- 3 From above, just anterior to the scapula and through the apex of the extrapleural space

The same technic is used as for aspirating the pleural cavity, penicillin is injected, a specimen sent for culture, and the final state checked by roentgenograms. A paradox pad is firmly reapplied after aspiration.

**Rupture of the Wound.** Rupture of the wound is rarely seen following thoracotomy, because an intact rib cage protects the muscles from the extremes of pressure of an explosive cough. In a thoracoplasty, however, when lengths of ribs have been



Fig. 10 Method of applying Paradox pad to stabilize the chest wall after thoracoplasty

removed, the wound is in direct contact with the explosive force of coughing, and, if unsupported, can rupture either at the upper or lower end. Excess of extrapleural fluid or air predisposes to rupture. To prevent rupture interrupted sutures are required, and the wound is held well supported by a physiotherapist, nurse, or doctor while the patient coughs. Immediately rupture of the wound occurs, fluid from the extrapleural space drains away.

If wound rupture is seen early, secondary suture after removal of the fluid and clot is required. If rupture is seen late, infection is certain, and adequate drainage through the axilla is required until the wound has healed and the extrapleural space has closed.

**Wound Infection and Extrapleural Space Infection.** WOUND INFECTION. Infection superficial to the muscle layer usually declares itself from 7 to 10 days after operation. Care in closing the wound and early removal of sutures tend to lessen the chance of this complication. Rarely it may be delayed as long as two months after operation when a nonabsorbent suture may cause a local abscess.

When infection is detected, the sutures are removed and the area is laid open and cleansed with dilute hydrogen peroxide irrigations and eusol packs thrice daily. When the wound has become cleaner, red lotion and ultraviolet irradiation will stimulate granulation tissue. If the amount of granulation tissue formed is large, secondary suture of the wound is indicated.

*Wound infection creates two problems*

- 1 Delay in performing the next stage of a thoracoplasty,
- 2 Infection of the extrapleural space

Although the infected area can be excised and the next stage of a thoracoplasty performed, nevertheless it is safer to have the wound soundly healed before operating and in this way avoid the risk of extrapleural space infection.

**EXTRAPLEURAL SPACE INFECTION.** This is usually traceable to some error in operating technic, operating-room ventilation, or an infection in the nasopharynx of some member of the operating team. It may also follow a superficial wound infection, a hematoma, or a ruptured wound. It also follows the rare rupture of a tuberculous cavity into the extrapleural space.

Extrapleural space infection is to be suspected when the temperature fails to subside in the second postoperative week, and when no other cause such as collapsed lower lobe or a pleural effusion is present. It is confirmed by diagnostic aspiration through the axilla with culturing of the organisms.

*Treatment* Infection usually responds to one of three methods of treatment. It may be treated by

- 1 Repeated aspiration,
- 2 Drainage of the extrapleural space, either by a Malecot catheter or by open axillary drainage,
- 3 Extrapleural space toilet and immediate resuture of the wound

*Aspiration* As the infection tends to persist in the retained blood clot that always collects after a thoracoplasty, it is unusual for aspiration and instillation of antibiotics to be adequate. With the more recent use of streptokinase and streptodornase to liquefy the blood clot, however, occasionally this treatment may prove completely effective.

*Drainage* This is most simply established through the axilla by means of a trocar cannula, Malecot catheter, and water-seal drain. Healing is encouraged by irrigating with eusol solution and connecting the drain to continuous suction. By the time the postoperative period of rest for the tuberculous lesion is completed, the sinus will have healed.

The diminishing size of the drained extrapleural space is checked by fortnightly sinograms. Finally, when the space has narrowed to a tube track, the tube which requires daily attention and which should reach to within three-quarters of an inch of the length of the sinus, is gradually shortened and finally removed.

If the sinogram shows that the extrapleural space has NOT been dependently drained by the Malecot catheter, then drainage must be improved by an operation through the axilla on the "letter-box" principle, and the space is allowed to heal by granulation.

*Extrapleural Space Toilet* When the thoracoplasty wound is healthy, an infected space is best treated by radical toilet which involves reopening the space posteriorly, evacuating all blood clot, cleansing with hydrogen peroxide and eusol solutions, and,

after final rinsing with saline leaving behind a solution of the appropriate antibiotic. The wound is then carefully resutured, and systemic antibiotic therapy is continued until the temperature has been normal for at least one week. Several postoperative aspirations of the space and instillation of antibiotics are required.

Formerly this complication was a hazard of any operation for pulmonary tuberculosis, but, with an appreciation of the value of prolonged chemotherapy both before and after operative treatment, this infection is now rarely seen.

## REFERENCES

- 1 Nissen, R. Extirpation eines ganzen Lungen flügels, *Zentralbl. Chir.*, 58 3003 1931
- 2 Haight, C. Total removal of left lung for bronchiectasis *Surg. Gynec. & Obst.*, 58 768 1934
- 3 Graham, E. A. and Singer J. J. Successful removal of entire lung for carcinoma of bronchus *J.A.M.A.* 101 1371 1933
- 4 Mason, G. A. Pneumonectomy—a preliminary report on two cases in which an entire lung has been successfully removed, *Brit. M. J.* 1 299 1935
- 5 ——— Extirpation of the lung, *Lancet*, 1 1047 1936
- 6 Edwards, A. T. Two cases of total pneumonectomy for bronchiectasis *Proc. Roy. Soc. Med.*, 29 221 1936
- 7 Roberts, J. E. H. Total pneumonectomy for bronchiectasis *Proc. Roy. Soc. Med.*, 29 220 1936
- 8 Griffin, S. G. Personal communication 1951
- 9 Dornie, J. Primary carcinoma of the bronchus. Prognosis following surgical resection (a clinico-pathological study of 200 patients) *Ann. Roy. Coll. Surgeons, England*, 10 165 1952.
- 10 Davies H. M. A provocative talk on pulmonary tuberculosis *Thorax*, 3 189 1948
- 11 Roberts, J. E. H. Lobectomy for bronchiectasis, *Proc. Roy. Soc. Med.* 29 220 1936
- 12 Susman, M. P. The surgical treatment of bronchiectasis *Australian & New Zealand J. Surg.* 6 249 1937
- 13 Churchill, E. D. and Belacy R. Segmental pneumonectomy in bronchiectasis. Lingula segment of left upper lobe *Ann. Surg.*, 109 481 1939
- 14 Overholt, R. H., and Woods, F. M. Prone position in thoracic surgery *J. Internat. Coll. Surg.* 10 216 1947
- 15 Brown, A. I. P. Posture in thoracic surgery *Thorax*, 3 161 1948.
- 16 Sellors, T. H. and Hickey M. D. Excision of the lung for pulmonary tuberculosis, *Thorax*, 4 82, 1949
- 17 Sauerbruch, F., and O'Shaughnessy L. *Thoracic Surgery* London, Edw. Arnold & Co., Ltd., 1937
- 18 Holst, J., Semb C., and Frimann-Dahl, J. On surgical treatment of pulmonary tuberculosis, *Acta, chir. Scandinav. Supp* 37 (act 1) 76 1 1935
- 19 Borrie J. The present state of surgery in the treatment of pulmonary tuberculosis, *New Zealand Med. J.*, 52 20 1953
- 20 Björk, V. O. Thoracoplasty—a new osteoplastic technique *J. Thoracic Surg.*, 28 194 1954
- 21 Brock, R. C. Osteoplastic thoracoplasty *Thorax*, 10 1 1955
- 22 Mason, G. A., in Sellors, T. H., and Livingstone, J. L. *Modern Practice in Tuberculosis*, London, Butterworth & Co. Ltd. 1952, vol. 2, p. 183
- 23 Hurt, R. L., and Bates, D. V. The value of quinidine in the prevention of cardiac arrhythmias after pulmonary resection, *Thorax* 15 39 1958

## INHALED FOREIGN BODIES

**Introduction.** Inhaled foreign bodies, especially in children under three years of age, are a real source of serious illness. They masquerade as acute laryngitis, acute asthma, pneumonia, lung abscess, bronchiectasis, or even empyema. Because such foreign bodies are often overlooked as a possible cause of disorder in those too young to speak for themselves, they carry an appreciable mortality. Robinson and Mushin (1) reported that in 1954 the total deaths from inhaled foreign bodies in England and Wales were as follows:

Pharynx and larynx	443
Digestive tract	29
Trachea, bronchus, and lung	11

Medicine is forever indebted to Chevalier Jackson for the lucid recording of his unparalleled experience in treating over 4,780 cases of foreign body in the air and food passages—on which he commenced publication in 1905 and summarized in his monograph of 1950 (2). He charted the pathology (3–6), physical signs (7–8), pitfalls and dangers, and pioneered many methods of extraction (9–10). Though the recent development of thoracic surgery has firmly established thoracotomy as an additional method of removal, time has proved the basic worth of Jackson's teaching.

There is at present no lessening of the incidence and variety of inhaled foreign bodies, yet were the rules of prevention observed, the incidence could be minimal. The important points are that.

1. The mouth was made for food not for pins, tacks, staples, needles, nails, small toys, and the like,
2. No child under six years of age should eat peanuts—only peanut butter,
3. No one should be thumped on the back while eating;
4. All small objects must be kept out of baby's reach. Babies require frequent correction and constant watching until their natural tendency to place everything in their mouths is overcome.

Despite all precautions, unattended infants have a propensity for inhaling peanuts, dried peas, apple skin, bone chips, eggshells, and kernels of corn. A sudden first attack of asthma in a baby must be regarded as evidence of an inhaled foreign body until proved otherwise; and, *since "seeing is believing," that means bronchoscopy*.

### PATHOLOGY

Inhaled foreign bodies occur largely in the young. In a personal four-year series of 24 cases, 20 were in children under 3 years of age. One of the most common offenders is the peanut, and 99 per cent of the cases occur in children (2). Though usually single, foreign bodies are occasionally multiple.

The right bronchus is most often affected because it follows the line of the trachea and is wider and the carina lies more to the left of the midline. Bronchial foreign bodies usually lodge in the main or lower lobe bronchi, Jackson found only 3 per cent in the middle lobe bronchus.

**Varying Effects of Foreign Bodies.** NATURE AND SITE. Inhaled foreign bodies are either *metallic* or *organic*. Their size and nature largely determine their pathologic effects in the lung. They may cause minimal partial or complete bronchial occlusion.

Small and smooth *metallic foreign bodies* such as pins that allow uninterrupted passage of air cause little immediate bronchial reaction. In time they oxidize and blacken, and they may penetrate the bronchial wall. *Partially obstructing foreign organic bodies* such as dried peas, beans, corn or peanuts, cause an intense local bronchitis which in time affects lung physiology. *Larger foreign bodies* metallic or organic, *totally occluding a bronchus* cause immediate atelectasis.

The effect on the patient and his respiratory system varies, depending on the site of lodgment of the foreign body.

*In the larynx* beside obstructing each phase of every breath, a foreign body rapidly causes laryngeal edema.

*In the trachea* if the foreign body is large, there is equal danger of total respiratory obstruction.

*In a main bronchus* usually the right, as the patient can still breathe with the unobstructed lung, there is less urgency therefore, the effects are either *immediate* or *delayed*.

**IMMEDIATE EFFECTS OF A BRONCHIAL FOREIGN BODY** The foreign body materially reduces ventilatory function by causing

- 1 Partial bronchial obstruction
- 2 Complete bronchial obstruction
- 3 A combination of both

**Partial Obstruction.** At first, air may pass freely in and out of the lungs—the so-called *symptomless interval* (Fig 1A). With mucosal reaction, however, there comes a time when air enters more easily than it leaves. This causes *obstructive emphysema* with overdistension of the affected lobe or lung, mediastinal displacement, and respiratory embarrassment (Fig 1B). Rarely this increased lobar pressure can even cause spontaneous pneumothorax from a ruptured bleb as seen in Chapter 1 Figure 5.

**Total Obstruction.** This may arise as soon as the foreign body is inhaled, or after a short period of obstructive emphysema, following which mucosal swelling and sputum retention finally block the bronchial lumen. Depending on the site of the foreign body the resulting atelectasis involves a lobe or the whole lung (Fig 1C).

**Combination of Partial and Total Obstruction.** This arises when the foreign body completely occludes one bronchial branch but only partially occludes the other. The blocked segment becomes atelectatic, while the partly occluded one becomes emphysematous (Fig 1D).





Fig 1A Normal chest roentgenogram in child with peanut at carina



Fig 1B Obstructive emphysema of right lung from peanut in right main bronchus



Fig. 1C. Inhaled pea causing total atelectasis of right lung and gross mediastinal displacement to right.

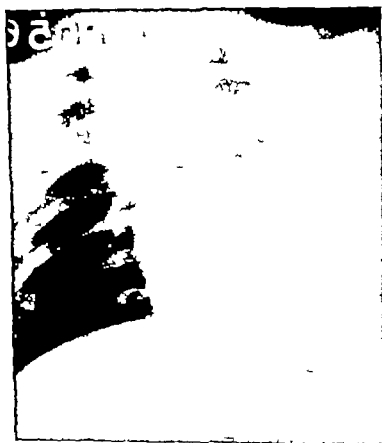


Fig. 1D. Cotyledon of pea causing right upper lobe atelectasis and lower lobe emphysema.

**DELAYED EFFECTS OF A BRONCHIAL FOREIGN BODY** When the foreign body is smooth and metallic, endobronchial changes are slow, taking possibly several weeks or months. When the object is organic, however, mucosal reaction is usually severe within a week, with all the sequelae of a blocked, infected bronchus. The lobe or lung becomes consolidated, bronchiectasis can develop in less than six weeks in the bronchi, and/or an abscess in the parenchyma may develop and be followed by pleural reaction and empyema (Fig 2A)

The foreign body itself may ulcerate through the bronchial mucosa or become embedded in exuberant granulations which progress to a fibrous stricture and simulate carcinoma (Fig 2B). Jackson and Jackson warn that the seeming exuberant granulation can, in fact, be carcinomatous fungations—a condition which they have seen on six occasions

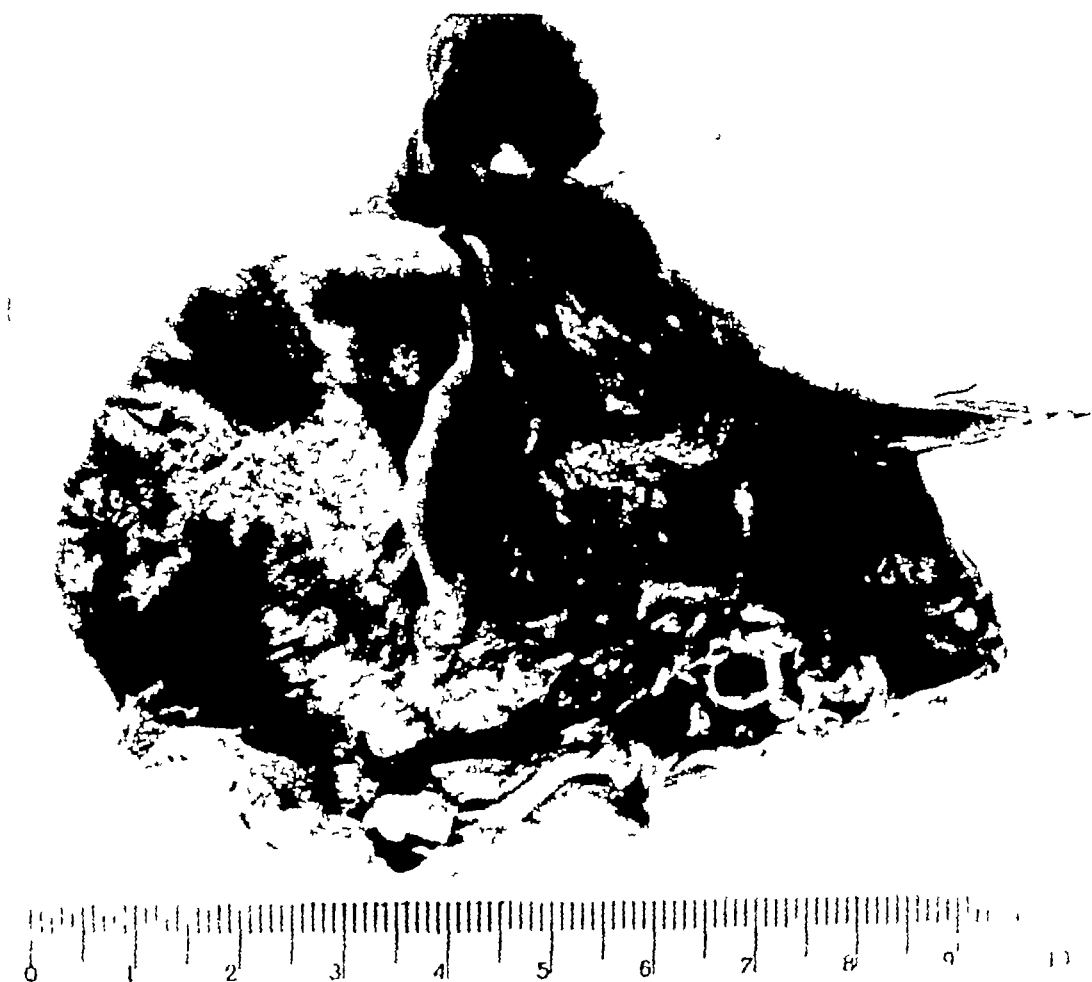


Fig 2A Lung of child who died on admission. She was believed for a month to have whooping cough. Actually, she had inhaled sprig of barley grass (*Bromus catharticus*) 4 cm x 0.75 cm (see point at right) which had caused widespread pneumonia and lung abscess, and had penetrated into right pleural cavity (Courtesy D. Perry)



Fig. 2B Lateral roentgenogram showing collapsed posterior basal segment, believed due to neoplasm, bronchoscopy showed small chop bone.

### CLINICAL FEATURES

These "range from no symptoms at all, to death by asphyxia, before medical aid can be obtained" (2) They vary with the site and mirror the pathologic state within the air passages

**Onset of Symptoms.** Almost invariably, there is a sudden episode of violent choking, coughing, cyanosis and dyspnea, often followed by a symptomless interval

On recovery an adult or older child will give a clear history of the accident. An infant, however cannot describe what has happened. He may have been alone at the time or he may have been playing with other children and been seen to choke. He may be discovered with a stidor or an asthmatic wheeze when circumstantial evidence such as a box of tacks or a partly eaten apple suggests the diagnosis. Practitioners and casualty officers should, therefore be chary of diagnosing "asthma" in young children before the possibility of a foreign body has been thoroughly excluded

Rarely, a foreign body may be inhaled without exciting any reactive reflexes at all. When, therefore, a child says he has inhaled a foreign body, even though clinically and roentgenologically nothing may be detected, his story must *not* be ignored, only bronchoscopy will relieve the anxiety of parents and practitioner alike.

**Intralaryngeal Foreign Body** The narrowness of the glottis and the danger of asphyxia make this a particularly hazardous location, for the object will usually be wedged fore and aft in the glottic opening, allowing respiratory exchange on either side. Alteration in the voice—crowing or hoarseness—and dyspnea may all be initial symptoms. In some, there is a latent interval for as much as three weeks (11) before edema of the glottis with acute respiratory obstruction inevitably develops.

**Intratracheal Foreign Body** The symptoms are the same as for intralaryngeal foreign body. Jackson described three pathognomonic signs:

- 1 An audible slap,
- 2 A palpatory thud,
- 3 An asthmatic wheeze

The first two are related to changing positions of the foreign body with each respiratory excursion, while the third reflects the degree of respiratory obstruction.

**Intrabronchial Foreign Body** Again, there are usually the symptoms of coughing, obstructed breathing, and wheeze. If there is no bronchial occlusion, there are no immediate signs or symptoms, and a latent period lasts until mucosal reaction and secretions obstruct air movement either partially or completely.

With *partial obstruction*, there are the signs of obstructive emphysema, with the trachea and mediastinum displaced away from the distended, hyperresonant lobe.

With *complete obstruction* and lobar or pulmonary atelectasis, the trachea, heart, and mediastinum are displaced to the affected side, with dullness to percussion and diminished or absent air entry.

**Investigations.** There are two important investigations, namely, roentgenography and bronchoscopy.

**ROENTGENOGRAPHY** This follows careful history taking and physical examination. It confirms the physical findings and may reveal:

- 1 The nature and position of the foreign body, if radiopaque,
- 2 The effect of the foreign body on bronchial mechanics, when partly or totally occluding a bronchus,
- 3 A normal x-ray film—when bronchial reaction and obstruction by an organic foreign body has not yet occurred.

Both posteroanterior and lateral films are required. In addition, when there is a possibility of an associated esophageal foreign body, the films should include the neck, abdomen, and pelvis. The importance of taking lateral roentgenograms including the larynx is shown in Figure 3, which concerns a baby 10 months old who choked while eating an egg sandwich. The fine vertical line of an intralaryngeal egg-shell was missed on the first anteroposterior roentgenograms but was noted three weeks later when the patient was admitted with acute edema of the glottis (11). An original lateral film would have shown it immediately as a plaque 1 cm square, and a dire emergency would thus have been avoided.

Typical x-ray films of nonmetallic foreign bodies are shown in Figure 1. In Figure 4, there was a metallic foreign body, and the diagnosis was obvious.

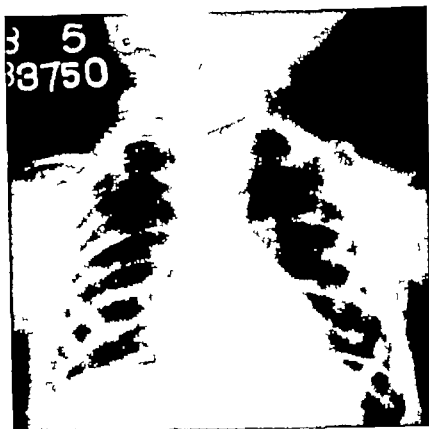


Fig. 3 Fine vertical line in glottis (opposite O) was all that anteroposterior roentgenogram revealed of intralaryngeal eggshell one cm. square (seen end on)



Fig. 4A. Screw head down at carinal level, not yet affecting bronchial mechanics. It was removed by bronchoscopy



Fig. 4B This patient was struck in the neck by flying metal bit which penetrated the trachea and fell into the posterior basal bronchus. Removed by bronchoscopy (Courtesy G Wooler)

**BRONCHOSCOPY.** When the roentgenograms are normal but there is a history or circumstantial evidence suggesting a foreign body, diagnostic and therapeutic bronchoscopy is required as being the only way of excluding and/or removing such a foreign body. In Figure 1A, though the chest film was normal, bronchoscopy revealed a peanut at the carina.

**Differential Diagnosis.** Any lesions causing, or caused by, obstructive emphysema or atelectasis, including spontaneous pneumothorax, unresolved pneumonia, bronchiectasis, lung abscess, neoplasm, and empyema must be considered. Such a list emphasizes how few lung disorders are primary lesions in themselves but rather are secondary to interference with the normal functioning of the bronchial system. All such lesions, therefore, must be bronchoscoped to exclude the possibility of a casual foreign body.

### TREATMENT

Once a foreign body is suspected or detected in the respiratory tree, it should be removed as soon as possible. The bronchial passages are essentially air ducts to the lung alveoli, and anything blocking them is fraught with the real danger of suffocation.

As only 3 per cent of foreign bodies may be coughed up, temporizing and hoping are useless. *There is one exception to this rule.* If several unsuccessful bronchoscopies have already been performed and the child is exhausted or has edema of the glottis, he must be resuscitated and possibly a tracheotomy performed before trying again.

#### TREATMENT AT THE MOMENT OF INHALATION

The time-honored practice of holding a child by the legs and banging his back is more likely to return the foreign body from bronchus to trachea, with the added danger of sticking at the larynx and asphyxiating the child. The best that can be hoped for is that, with the child standing upright, the foreign body is drawn into and lodges in one of the main bronchi, thus allowing normal respiration with the other lung. In practice, this usually happens.

#### TREATMENT FOR FOREIGN BODY IN BRONCHUS

**Formal Removal.** From a surgical standpoint, this is a serious operation, requiring the careful attention of a team consisting of a skilled bronchoscopist and anesthesiologist. The problem is carefully considered, and the kind of foreign body is discussed. If necessary, similar records are reviewed and practice removal carried out with use of a wide-bore rubber tube as a model trachea.

**Anesthesia.** In infants, the author favors the use of open ether preceded by a suitable dose of subcutaneous atropine. This method causes no respiratory depression and usually allows adequate relaxation and time for removal during the phase of recovery from the anesthetic. The bronchoscope is lighted from a battery source which limits the current and safeguards against explosion in the event of a short circuit.

In older children, a sleep dose of thiopentone followed by adequate spraying of the vocal cords and trachea with local anesthetic proves safe and satisfactory. Relaxants must not be used; they are dangerous, because, in their presence, adequate ventilation of the lungs during bronchoscopy is difficult.

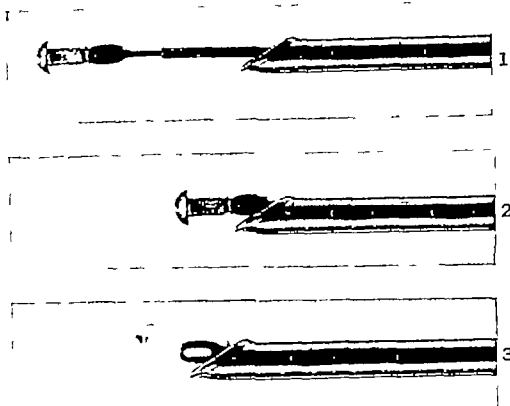


Fig. 5 1 Method of extracting screw from bronchial tube. 2, With the foreign body first drawn into the mouth of the bronchoscope, bronchoscopy forceps and foreign body are withdrawn in one steady movement. 3 Method of extracting peanut from bronchial tree, same steps as above.

*In adults* the usual local anesthesia for diagnostic bronchoscopy—for example subcutaneous premedication of omnopon 20 mg and scopolamine 0.43 mg. an hour beforehand, followed by an amethocaine lozenge (50 mg) and painting the throat and glottis with local anesthetic—is usually sufficient. If general anesthesia is required for the apprehensive patient, the procedure is similar to that for children, i.e. full local anesthesia carried out under a dose of thiopentone.

#### INTRALARYNGEAL FOREIGN BODY

**Uncomplicated.** Intralaryngeal foreign bodies usually occur in infants and very often are associated with incipient edema of the glottis. With the child anesthetized and placed in the head-down position, the bronchoscope is passed, the foreign body visualized and gently grasped on its presenting point and withdrawn. The author favors Jackson's 3 mm. light weight peanut forceps.

**AFTER-CARE.** The child is returned to the ward and nursed in a Croupette (see Chapter 2 Fig. 16) which gives a fine mist of nebulized water vapor. To this may be added nebulized detergent, e.g. sodium lauryl sulfate to reduce the surface tension of any endobronchial secretions. The child requires strict observation by a special nurse and should be kept in hospital for at least three days after removal of the foreign body to avoid the real possibility of delayed edema of the glottis.

**With Edema of the Glottis.** The foreign body is removed as in the uncomplicated case but the bronchoscope should be immediately reinserted into the trachea to relieve the obstruction, allow normal respiratory excursion, and give time for prompt formal tracheotomy. Thereafter the same after-care is observed.



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**Differential Diagnosis.** Any lesions causing, or caused by, obstructive emphysema or atelectasis, including spontaneous pneumothorax, unresolved pneumonia, bronchiectasis, lung abscess, neoplasm, and empyema must be considered. Such a list emphasizes how few lung disorders are primary lesions in themselves but rather are secondary to interference with the normal functioning of the bronchial system. All such lesions, therefore, must be bronchoscoped to exclude the possibility of a casual foreign body.

### TREATMENT

Once a foreign body is suspected or detected in the respiratory tree, it should be removed as soon as possible. The bronchial passages are essentially air ducts to the lung alveoli, and anything blocking them is fraught with the real danger of suffocation.

As only 3 per cent of foreign bodies may be coughed up, temporizing and hoping are useless. *There is one exception to this rule.* If several unsuccessful bronchoscopies have already been performed and the child is exhausted or has edema of the glottis, he must be resuscitated and possibly a tracheotomy performed before trying again.

#### TREATMENT AT THE MOMENT OF INHALATION

The time-honored practice of holding a child by the legs and banging his back is more likely to return the foreign body from bronchus to trachea, with the added danger of sticking at the larynx and asphyxiating the child. The best that can be hoped for is that, with the child standing upright, the foreign body is drawn into and lodges in one of the main bronchi, thus allowing normal respiration with the other lung. In practice, this usually happens.

#### TREATMENT FOR FOREIGN BODY IN BRONCHUS

**Formal Removal.** From a surgical standpoint, this is a serious operation, requiring the careful attention of a team consisting of a skilled bronchoscopist and anesthetist. The problem is carefully considered, and the kind of foreign body is discussed. If necessary, similar records are reviewed and practice removal carried out with use of a wide-bore rubber tube as a model trachea.

**Anesthesia.** *In infants*, the author favors the use of open ether preceded by a suitable dose of subcutaneous atropine. This method causes no respiratory depression and usually allows adequate relaxation and time for removal during the phase of recovery from the anesthetic. The bronchoscope is lighted from a battery source which limits the current and safeguards against explosion in the event of a short circuit.

*In older children*, a sleep dose of thiopentone followed by adequate spraying of the vocal cords and trachea with local anesthetic proves safe and satisfactory. Relaxants must not be used, they are dangerous, because, in their presence, adequate ventilation of the lungs during bronchoscopy is difficult.

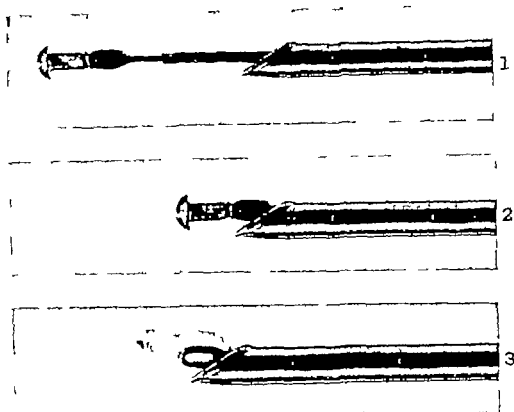


Fig. 5 1 Method of extracting screw from bronchial tube. 2, With the foreign body first drawn into the mouth of the bronchoscope, bronchoscopy forceps and foreign body are withdrawn in one steady movement. 3 Method of extracting peanut from bronchial tree same steps as above.

*In adults* the usual local anesthesia for diagnostic bronchoscopy—for example subcutaneous premedication of omnopon 20 mg. and scopolamine 0.43 mg. an hour beforehand, followed by an amethocaine lozenge (50 mg.) and painting the throat and glottis with local anesthetic—is usually sufficient. If general anesthesia is required for the apprehensive patient, the procedure is similar to that for children, i.e. full local anesthesia carried out under a dose of thiopentone.

#### INTRALARYNGEAL FOREIGN BODY

**Uncomplicated.** Intralaryngeal foreign bodies usually occur in infants and very often are associated with incipient edema of the glottis. With the child anesthetized and placed in the head-down position, the bronchoscope is passed, the foreign body visualized and gently grasped on its presenting point and withdrawn. The author favors Jackson's 3 mm. light weight peanut forceps.

**AFTER-CARE.** The child is returned to the ward and nursed in a Croupette (see Chapter 2, Fig. 16) which gives a fine mist of nebulized water vapor. To this may be added nebulized detergent, e.g. sodium lauryl sulfate, to reduce the surface tension of any endobronchial secretions. The child requires strict observation by a special nurse and should be kept in hospital for at least three days after removal of the foreign body to avoid the real possibility of delayed edema of the glottis.

**With Edema of the Glottis.** The foreign body is removed as in the uncomplicated case, but the bronchoscope should be immediately reinserted into the trachea to relieve the obstruction, allow normal respiratory excursion, and give time for post-operative tracheotomy. Thereafter the same after-care is observed.

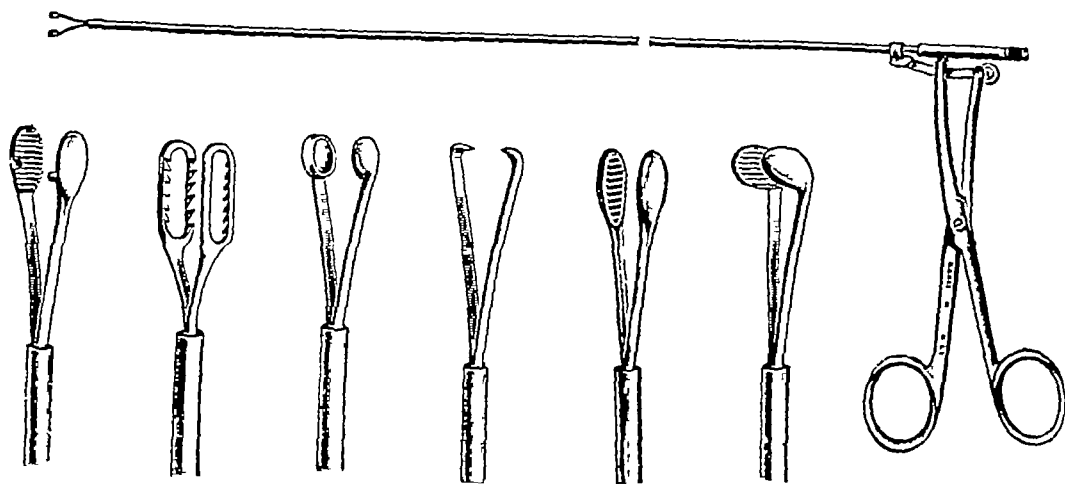


Fig 6 Selection of forceps for removing foreign bodies from bronchial tree (Courtesy Genito-Urinary Mfg Co Ltd, London)

### INTRATRACHEAL AND INTRABRONCHIAL FOREIGN BODIES

The bronchoscope is gently passed and advanced to the carina. If there is bronchospasm, it can be relieved with two or three drops of a local anesthetic solution. If the spasm is severe, the bronchoscope is best withdrawn a little and the patient given oxygen down the side tube. First the carina is inspected, then the right main bronchus.

**Organic Foreign Bodies.** If the foreign body is half a cotyledon of a peanut, the surrounding bronchial wall may be found in spasm. This can be overcome by gently insinuating the beak of the bronchoscope toward the peanut, but the bronchoscope, when infant size, is rarely large enough to accommodate the nut.

The peanut forceps is inserted, opened, a blade passed on either side, and the instrument tightly closed. The peanut is then drawn into the mouth of the bronchoscope, the forceps is firmly held and steadied against the bronchoscope, and peanut, forceps, and bronchoscope are removed in one steady movement (Fig 5). *The first chance is the best chance.*

**THE DROPPED FOREIGN BODY** If the foreign body is not steadied in the mouth of the bronchoscope, it will become detached, usually in the trachea or at the glottis, and cause respiratory obstruction. The bronchoscope must immediately be reinserted and the foreign body regrasped. If respiratory obstruction is acute, the foreign body should be pushed temporarily into one bronchus and the bronchoscope kept in as an airway. If the foreign body is small, it may have been aspirated into the opposite bronchial tree, as the inspiratory column of air will still be greater to that side.

When the foreign body is a dried pea, bronchial secretions cause it to swell and produce complete atelectasis of a lung (Fig. 1 C). Removal may then be done in three parts—first the skin, then each of the cotyledons.

As multiple foreign bodies can and do occur, it is wise to check the bronchial tree after seemingly successful removal. Thereafter, the bronchoscope is removed, the patient turned on his side, and oxygen given to overcome any laryngeal spasm. Only when breathing is free and steady is the patient returned to the ward.

If no foreign body is found in the bronchus but there is a clear history of ingestion and even signs of collapse of one lung (usually the right), esophagoscopy is performed, for the foreign body may in fact be in the esophagus, pressing forward on the right main bronchus (see Chapter 14).



Fig 7 Needle that broke and penetrated into bullous cyst in left upper lobe removed by thoracotomy

**Metallic Foreign Bodies.** Though there is less danger of severe reaction, early removal is still necessary for there is no knowing when total bronchial occlusion will occur. If original chest films have been taken and the patient has thereafter traveled some distance for treatment, further films are necessary in case the foreign body has changed its position.

**BRONCHOSCOPIC REMOVAL** Centrally placed pins, brass-headed upholstery tacks, carpet tacks, nails, screws, tubes, hairpins, safety pins, staples and the like all require removal by the special and careful technics described by Jackson and Jackson (2) (Fig. 6). When unfavorably placed, they may require rotation to make the presentation easier. It may be possible to bend a pin into the mouth of the bronchoscope to close an open safety pin with a Clerf safety pin extractor, to close a hairpin with a double pointed foreign body forceps, or to insinuate the blades of the expanding bronchoscope around a staple.

**THORACOTOMY** When the foreign body is hopelessly impacted or is lying in a peripheral bronchus and is a pin, needle, or small nail, then it is far less hazardous and far better practice to stop instrumentation and remove it by thoracotomy and bronchotomy. The affected bronchus is located and dissected free, the foreign body felt, a small incision made, and the object removed. The bronchial wall is then closed with fine silk sutures and the chest wall closed over water seal drainage. If the segment or lobe of lung beyond is irrecoverably collapsed, infected, and bronchiectatic, it should be resected at the same time.

Similarly, for foreign bodies penetrating into the lung parenchyma (e.g., needles or shot) there are now no grounds for using the Jackson method of thoracopuncture whereby forceps were introduced from outside under local anesthesia, and guided to the foreign body under x-ray control. The object was then firmly grasped

and extracted. Such a method ignores the fibrous tissue in which many of these foreign bodies may lie, the surrounding blood vessels, and the certain chance of pneumothorax and possible hemothorax. With thoracotomy now a safe procedure, this alone is the logical and acceptable means of treating such cases (Fig. 7)

#### RULES FOR SUCCESSFUL ENDOSCOPIC REMOVAL OF FOREIGN BODIES

1 After all clinical and roentgenographic evidence has been carefully sifted, there should be one and only one well-planned attempt at removal. A preliminary "look" cannot be entertained

2 Check the bronchoscope light, suction, and, above all, the forceps, seeing that the jaws grip firmly when closed.

3 Advance the bronchoscope carefully toward the foreign body, gently sucking away surrounding mucus. Do not touch tissue with the suction point lest it excite hemorrhage

4 Inspect the foreign body, note its characteristics and the most suitable place for grasping it. With screws, nails, pins and so on, always seek the point (Fig. 6b)

5 See that the bronchoscope and the axis of the foreign body are in one and the same straight line

6 Gently advance the forceps down the bronchoscope, open the points, advance them around the chosen part of the object, and close carefully and firmly

7 Now, bring the point of the foreign body into the mouth of the bronchoscope. With the bronchoscope, forceps, and point of the foreign body as one unit, remove from the trachea

8 The mortality from failing to remove a foreign body can *never justify violence during removal, which itself may prove fatal*

9 Be prepared to do an esophagoscopy, for a foreign body may drop into the pharynx and be swallowed. As has been noted, pulmonary signs may be due, not to an endobronchial foreign body, but to one in the esophagus

10 If breathing becomes difficult, withdraw the bronchoscope a little, but do not remove it entirely, for it is an ideal airway allowing emergency mouth-to-bronchoscope insufflation of both lungs and subsequent oxygen therapy

11 If the foreign body cannot be removed *endoscopically* from a baby within 15 minutes, or from a child in 25 minutes, then desist, and assess the situation to determine whether open operation should not be performed instead. There is no virtue in "prolonging the agony," and the chances of glottic edema become great. In the experience of Jackson and Jackson (2), 99 per cent of such foreign bodies are removable by endoscopy.

#### POSTOPERATIVE CARE AND COMPLICATIONS

After withdrawal of the bronchoscope, the child is turned on his side, and any gagging is overcome with a few breaths of pure oxygen

The immediate danger from bronchoscopy is *edema of the glottis*, which can be delayed for several days. The child should be nursed in hospital and kept quiet. If there is the slightest evidence of advancing hoarseness, he should immediately be placed in a Croupette with nebulized water vapor and detergent as for laryngeal foreign body

If laryngeal obstruction increases, tracheotomy becomes necessary. It should not



# Esophageal Emergencies

## 14

### FOREIGN BODIES IN THE ESOPHAGUS

Foreign bodies in the esophagus are doubly dangerous, for there are risks both from their impaction and from their extraction. It may be thought that, with fluoroscopy and esophagoscopy, they have lost all their terrors, but the occurrence of an occasional tragedy emphasizes the fact that these are still serious emergencies demanding the utmost respect. Furthermore, with all foreign bodies the question arises, "Is there any local esophageal lesion that has caused its arrest, and, if so, what is its nature?" This question must always be kept in mind, and if organic disease is discovered, it should be further investigated and treated.

#### **PATHOLOGY**

As Grey Turner (1) taught, the most important events in the natural history of esophageal foreign bodies are

- 1 They may pass safely,
- 2 They may pass, but leave a trail of damage in their wake—transit injuries,
- 3 They may become impacted with rapidly fatal consequences,
- 4 Impaction may be followed by slow erosion, eventually ending in death,
- 5 In cases that survive, stricture or fistula may follow.

**Varieties of Esophageal Foreign Bodies.** Esophageal foreign bodies fall into two groups:

- 1 The *radiotranslucent*—usually consisting of organic matter,
- 2 The *radiopaque*—usually metallic in nature.

Of the first variety, partly chewed food such as meat, fish, gristle, fruit skins, peas, and beans are commonly recovered. Of the second variety, pieces of animal and fish bones, fruit stones, parts of dentures, straight pins and safety pins, bottle tops, razor blades, and so on have all been removed from patients.

The ingestion of most foreign bodies is usually an accident associated with careless eating and failure to masticate properly. The elderly are most susceptible, especially those with dentures or the edentulous who attempt to swallow half-chewed food. Younger adults, however, may bolt too big a bolus, as shown in Figure 1, or they may swallow all sorts of objects for a wager, as shown in Figure 2. If a foreign body passes the cardia, it will probably pass safely through the rest of the alimentary tract, though the time required may vary from 30 hours up to several weeks.

**Pathologic Lesions and Site of Impaction.** In most cases involving foreign bodies, the esophagus is anatomically normal, but the *possibility of a pathologic lesion* must always be kept in mind. In infants, such a lesion is most commonly an esophageal stricture from reflux esophagitis (Fig. 3). The narrowed lumen at the stricture becomes completely blocked when solids are first taken, as shown in Figure 3. However, the blockage may occur at a congenital web stricture at the level of the aortic arch. An old caustic scar stricture can become blocked by food taken carelessly in



1 2 3 4 5  
CMS

Fig. 1 Meat from large chop swallowed unchewed, and impacted in the cervical esophagus.



Fig. 2 Bottle top impacted in esophagus; two others reached stomach and were passed naturally in seven days.



Fig. 3A Reflux esophagitis stricture totally blocked by pith of an orange in child, aged 15 months.



Fig. 3B Post-removal esophagram showing the stricture



a moment of forgetfulness. In the later decades of life, the lesion may be cardiospasm (Fig 4), reflux esophagitis stricture, or neoplasm.

The *site of impaction* varies with the size and shape of the foreign body, being most common just below the cricopharyngeal sphincter, at the level of the aortic arch, or just above the cardia. No site, however, is immune.

**Complications with a Foreign Body.** Usual complications are two in number: perforation and ulceration.

**PERFORATION** Sharp foreign bodies such as a fish bone may quickly perforate through the esophageal wall and cause acute mediastinitis, pericarditis, or empyema. Rarely, the aorta may be perforated. Unless these acutely perforating foreign bodies are recognized and treated early, they will prove fatal. Occasionally, they are surprisingly harmless. Sellors (2) records how a child aged three was found with a razor blade in his esophagus.

A foreign body may be responsible for two other forms of esophageal perforation:

- 1 From the esophagoscope, if it is passed hastily and without due care,
- 2 From the foreign body itself, if it is removed too vigorously.

In both instances, the tear must be recognized and treated promptly if life is to be preserved (see Chapter 19).

**ULCERATION** In time, the esophageal mucosa becomes ulcerated, leading to stricture formation. Rarely, a foreign body with smooth surfaces, such as a coin, may remain in the esophagus for years, slowly ulcerating into the aorta (3) or into a bronchus to produce a bronchoesophageal fistula, pulmonary sepsis, empyema, and external fistulas (4). Though Jackson and Jackson (2) record how 1 patient



Fig 4 Large lumps of partly-chewed food caused total dysphagia in this patient with cardiospasm; relief followed removal by esophagoscopy. Film taken after disimpaction.

had a wooden button lodged in the esophagus and causing chronic dysphagia for 17 years, Thorek (5) concludes from clinical data that patients with overlooked foreign bodies almost never survive longer than 5 or 6 years and usually succumb within a year

### CLINICAL FEATURES

**Symptoms.** Almost invariably, *children or adults* are conscious of having swallowed a foreign body and can give a clear history of choking or gagging. There is usually a localized sensation of discomfort and possibly pain, especially if the object is large and sharp. Each act of swallowing aggravates the symptoms. If a foreign body becomes dislodged and passes down the alimentary canal, symptoms of local pain may persist for several days, especially if the esophageal wall has been injured during the transit of the foreign body. With *infants* however, no history can be obtained. The child may cry with each swallow or regurgitate everything when blockage is total.

*The history is most important and must be believed.* One child who three months previously had swallowed something from his coat pocket believing it to be a candy, developed chronic cough and lost weight. Repeated roentgenograms had shown nothing, but esophagoscopy revealed the cause—a radiotranslucent button wedged in the esophagus and pressing on the right main bronchus.



Fig. 5 Fruit stone impacted in cervical esophagus

**Physical Signs.** These are few. The appearance of the patient may belie the seriousness of the condition. Watching the patient swallow will reveal whether he hesitates or winces, both confirming his complaint of dysphagia. If he spontaneously retches up blood, there has been mucosal injury.

Foreign bodies in the upper third of the esophagus usually produce local tenderness. If there is cervical induration or surgical emphysema, it means that the foreign body has perforated the esophagus and produced acute mediastinitis or cervical cellulitis. Similarly, the signs of empyema or acute pericarditis indicate that it has ruptured into these serous cavities.

**Investigations.** There are only two important investigations:

- 1 Roentgenograms, including a barium swallow;
- 2 Esophagoscopy.

These are complementary examinations. The first does not exclude the second or lessen the need for it.

**ROENTGENOGRAMS.** Large organic foreign bodies may be outlined by a barium swallow, but small ones (including small pieces of bone) may escape x-ray detection.

Posteroanterior and lateral esophageal roentgenograms are of value in detecting radiopaque foreign bodies as in Figure 5. Again, when a modern radiotransparent acrylic dental plate has been repaired with wire or has wire incorporated in its substance, roentgenograms confirm the presence and level of the foreign body (6) (see Figs 6A and B).

When, however, the esophagus has been perforated, there will be the characteristic evidence of mediastinal emphysema or empyema. When a metallic foreign body has passed on into the stomach, serial x-ray films are of value in checking progress through the alimentary canal.

**ESOPHAGOSCOPY.** Like bronchoscopy for a bronchial foreign body, esophagoscopy is the most important method of treating esophageal foreign bodies. It should not be undertaken until everything is in readiness for removal. It is indicated for all cases of foreign body known, or suspected to be, in the esophagus. See below for details of this procedure.

## MANAGEMENT

For management, foreign bodies fall into three groups.

- 1 Those which reach the stomach;
- 2 Those impacted in the esophagus,
- 3 Those which perforate and produce complications.

### FOREIGN BODIES REACHING THE STOMACH

These merely require observation. Even needles and pins can pass naturally. In the absence of any local pain or tenderness, no further action is required. If it is decided to perform a laparotomy, roentgenograms must be taken on the way to the operating room to check that the foreign body has not moved on.

### FOREIGN BODIES IMPACTED IN THE ESOPHAGUS

These require removal by esophagoscopy, but, as Jackson and Jackson point out, "should the patient be in a bad condition from previous ill-advised or blind attempts



Fig 6A. Radiotransparent dental plate previously repaired with piece of wire which alone showed on x ray film.

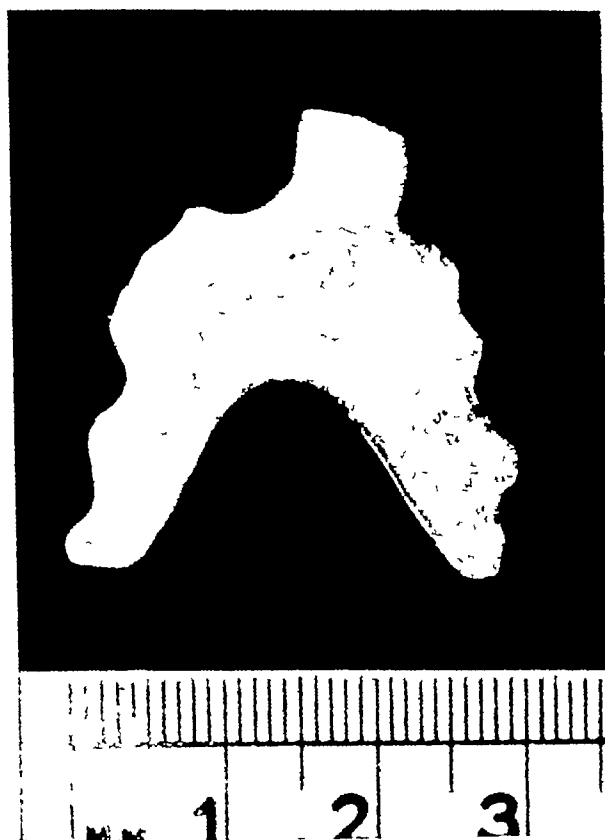


Fig 6B Recovered dental plate

at extraction, endoscopy should be delayed until the traumatic esophagitis has subsided and the general state improved "

**Anesthesia.** General anesthesia is desirable. In infants, single-dose ether anesthesia followed by intubation is usually sufficient. In older children and adults, the patient is intubated after a sleep dose of thiopentone and suxamethonium as relaxant. During the ensuing period of relaxation, the patient's lungs must be continuously ventilated and the esophagoscope passed.

**Esophagoscopy.** As already mentioned, due care must be observed while the instrument is being introduced. For example, in one referred case there was a history of struggling to introduce the esophagoscope into a patient under light nitrous oxide anesthesia, followed by a sudden easing of resistance and dramatic gushing of air with each breath. Investigation confirmed that the esophagoscope had perforated the right vallecula and had plunged into the right pleural cavity.

With the patient relaxed, therefore, and his chin forward, the esophagoscope is placed just above the cricopharyngeal sphincter. This sphincter is always closed except for swallowing, vomiting, or belching. An obturator—preferably a No. 18–20 French esophageal bougie—is insinuated through the sphincter, and thereafter the esophagoscope follows.

The esophageal lumen is carefully inspected, fluid aspirated, and the patient's head adjusted on the headrest as the esophagoscope advances toward the cardia. Some narrow foreign bodies, such as a thin piece of bone or a coin, may be inadvertently bypassed and recognized only as the esophagoscope is withdrawn.

**THE REMOVAL.** When the foreign body is detected, the same rules apply as for extraction from the bronchial tree. Inspect the foreign body, note its character and the most suitable place for grasping it. When it is small—e.g. a spicule of bone—it will readily be drawn into the lumen of the esophagoscope. When it is larger—e.g. a coin

## Management

or part of a dental plate—it is grasped and drawn to the mouth of the esophagoscope. Then, with esophagoscope forceps and foreign body held in one firm grip the whole is withdrawn as a unit. The dangerous area is the cricopharyngeal sphincter where mucosal tearing can occur. This danger, however, is greatly lessened by the maneuver of having the foreign body trail in continuity with the esophagoscope.

Some foreign bodies, such as razor blades, present special problems. Horowitz (7) was able to snip one at either end and remove it safely in two parts. The mechanical problems of removal of safety pins, straight pins, buttons, can-opening keys, studs and so on are described in detail by Jackson and Jackson (2). For upward-pointing open safety pins, Jackson pioneered "endogastric version" (8) in which the pin is seized at the spring by a ring forceps and, with the point trailing behind, is pushed on into the lumen of the stomach. The pin is then grasped and withdrawn, and it automatically becomes rotated and is thereafter removed as described above with minimal danger of trauma. In an alternative method, the point is suitably sheathed by bringing it into the esophagoscope and thereafter the esophagoscope forceps and pin are gently removed. Still another method employs Clerf's safety pin closer.

Pin button-badges in the esophagus must be grasped opposite the pin point so that the point trails harmlessly behind.

If a can-opening key is presenting with the point uppermost, this is grasped, drawn into the esophagoscope and the key withdrawn safely.

In general it may be said that, if the point that could otherwise tear the esophageal wall can be drawn into the esophagoscope, safe removal is assured. If this is not possible, then the foreign body must be grasped in such a way that the point trails harmlessly behind.

However, when the foreign body is tightly impacted and cannot easily be broken with shears and when there is real danger of perforating the esophageal wall during manipulation, then the esophagoscope must be removed and the patient made ready for open operation and esophagotomy.

**Cervical Esophagotomy.** This is indicated when a foreign body such as a partial denture with many sharp hooks lies wedged in the esophagus above the thoracic inlet.

A three inch incision is made along the anterior border of the right sternomastoid muscle, the muscle and carotid sheath are retracted laterally and the trachea and esophagus are retracted medially. The middle thyroid veins are divided, and thereafter the esophagus is isolated. If the foreign body is easily palpated, a vertical incision is made in the esophageal wall and the object removed. Even at this stage it may need to be broken with bone shears before it is finally extracted. The esophageal wall is sutured in two layers with interrupted chromic catgut, and the wound is closed over a corrugated rubber drain, using interrupted catgut for the subcutaneous layer and nylon for the skin.

**AFTER-CARE.** Since he now has a potentially infected cervical wound, the patient is given a short course of prophylactic antibiotic therapy. He can take water by mouth the first day, advancing to a general fluid diet for the next 10 days, and thereafter to a normal diet. The drain is removed on the second postoperative day and the wound is usually well closed by the tenth day. Residual scarring is minimal.

**Trans thoracic Esophagotomy.** When the foreign body is firmly wedged in the thoracic esophagus, exploration must be done—preferably from the right hand side which gives easy access to its entire length. Depending on the position of the fore-

body, the chest is opened through the fourth, sixth, or eighth rib bed. The lung is retracted, the mediastinal pleura vertically incised, the arching azygos vein divided if required, and the esophagus isolated. A tape or catheter sling is passed around it, a vertical incision made over the foreign body which is then extracted, and the opening is closed in two layers with interrupted chromic catgut sutures. The chest wall is closed over water-seal drainage.

**AFTER-CARE** This follows the same line as for cervical esophagotomy and routine thoracotomy. Roentgenograms are taken the day after an operation and, if the lung is fully expanded, the tube is removed. Diet is as described above, but due care must be taken, for it is possible for such an esophageal wound to leak and cause an empyema. Skin sutures are removed after a week, and shortly thereafter the patient may be discharged as convalescent.

Sellors (2) records a successful right fourth rib thoracotomy on a three-year-old boy when earlier esophagoscopy had failed to remove a razor blade firmly wedged in congested mucosa of the midesophagus. Similarly, Grey Turner (9) removed by transthoracic esophagotomy a tooth plate impacted for 15 years.

#### FOREIGN BODIES PERFORATING AND PRODUCING COMPLICATIONS

A foreign body may perforate

- 1 The *cervical esophagus* and enter the soft tissues of the neck,
- 2 The *thoracic esophagus* and enter the mediastinum, pleural or pericardial cavities

When the perforation is promptly recognized before infection has occurred, treatment follows that described in Chapter 19 under esophageal perforation. When, however, infection has already occurred, depending on the site of perforation and the direction of spread of the foreign body, there will be acute cervical cellulitis, mediastinitis, empyema, or suppurative pericarditis, each of which is described in the appropriate chapter.

In conclusion, while most foreign bodies are successfully removed by esophagoscopy, some, because of infection or complications, require emergency operation. This should not be delayed, once the true state of affairs is appreciated.

#### REFERENCES

- 1 Turner, G. G. *Injuries and Diseases of the Oesophagus*, London, Cassell and Co., Ltd., 1946
- 2 Sellors, T. H. Razor blade in esophagus—transthoracic removal, *Brit J Surg*, 34:276, 1947
- 3 Turner, G. G. Death from perforation of the aorta by a halfpenny impacted in the esophagus, *Lancet*, 1:1335, 1910
- 4 Jackson, C., and Jackson, C. L. *Bronchoesophagology*, Philadelphia, W. B. Saunders Co., 1950
- 5 Thorek, P. *Diseases of the Esophagus*, Philadelphia, J. B. Lippincott Co., 1952
- 6 Borrie, J., and Donaldson, K. I. Partial denture in oesophagus, *New Zealand Dental J*, 49:237, 1953
- 7 Horowitz, S. Personal communication
- 8 Jackson, C. Esophagoscopy removal of open safety pin, by a new method, *Laryngoscope*, 20:446, 1910
- 9 Turner, G. G. Tooth plate impacted in gullet for fifteen years, removal by transthoracic esophagotomy, *Brit J Surg*, 34:290, 1947

## CONGENITAL ESOPHAGEAL EMERGENCIES TRACHEOESOPHAGEAL FISTULA AND ATRESIA

**Historical Note.** Franklin (1) states that congenital esophageal lesions were described by Gibson (2) Physician General to the Army in 1697 and that MacKenzie (3) in 1884 collected records of 43 cases. The most common variety, blind upper esophageal pouch and distal pouch communicating with the trachea, was fully described by Keith (4) in 1910.

Regarding surgical treatment using staged antethoracic reconstruction Ladd (5) reported five successes in 1939 and Leven (6) another in 1940. The ideal, however of primary esophageal anastomosis was attempted by Lanman (7) in 1936 on four patients all of whom died. In 1939, Shaw's (8) patient survived 12 days. In 1941 Haight (9) first successfully performed primary end-to-end esophageal anastomosis.

**Embryology** Normally, during the fourth week of intrauterine life two longitudinal ridges appear on the lateral walls of the developing foregut. Between the fifth and sixth weeks these proliferating ridges fuse from the region of the lung bud forward to the head and thereby separate the esophagus from the larynx and trachea. With further cellular proliferation, both structures become considerably narrowed though not occluded and finally recanalize into their definitive form. However oblique fusion of these ridges or failure of esophageal recanalization results in

- 1 Esophageal atresia with tracheoesophageal fistula
- 2 Atresia without tracheoesophageal fistula
- 3 Tracheoesophageal fistula without atresia.

Of these the first group is the most common.

### PATHOLOGY

Regarding incidence while Franklin (10) records it as 1 in 2,500 births Belsey and Donnison (11) found it was greater than 1 in 800 births.

Various classifications have been put forward by Vogt (12) and Swenson (13) but for practical purposes all these lesions fall into the three groups already mentioned. Swenson's five types are

**TYPE I** Rare. The proximal esophagus ends as a blind superior mediastinal pouch and the distal esophagus begins as a blind posterior mediastinal segment—3 per cent.

**TYPE II** The most common. There is a blind proximal pouch but a fistula between the trachea and distal esophagus—90 per cent.

**TYPE III** Rare. There is a blind distal segment, but a fistula between the proximal pouch and trachea.

**TYPE IV** Rare. There is a double fistula from trachea to each esophageal segment.

**TYPE V** Rare. There is tracheoesophageal fistula without atresia.

To these five might be added

**TYPE VI** Esophageal atresia at the level of the tracheal bifurcation without fistula as described by the writer (14) (Fig. 1).



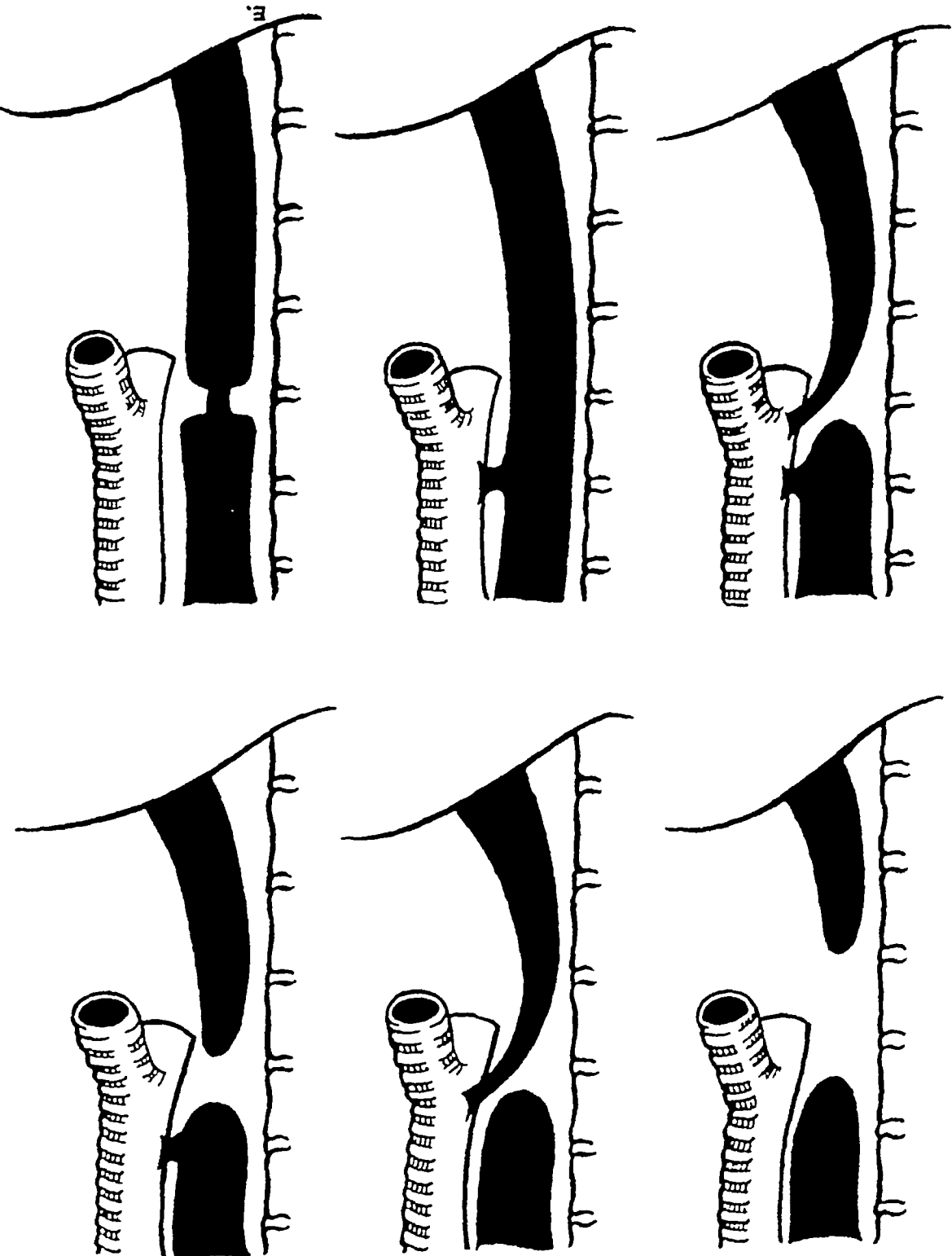


Fig 1 Six types of atresia of esophagus and tracheoesophageal fistula

Type I	Type II	Type III
Type IV	Type V	Type VI

## Clinical Features

**Atresia with Fistula.** In Type II, the upper blind pouch is uniformly dilated and hypertrophied, but as Shaw and others (15) point out, it may vary in length from a small pouch high in the thorax to a blind segment extending almost to the diaphragm. Similarly, the lower esophagus may be well developed and enter the posterior tracheal wall with a fistulous opening as high as 2 cm above the tracheal bifurcation, or may be a small poorly developed muscular tube, tapering to form a long, narrow fistula which opens at the tracheal bifurcation or into one or other of the main bronchi.

Thus the two segments may overlap by 2 to 3 cm and at first sight may appear normal from without, or they may be unduly separated and present little hope of primary anastomosis.

**Esophageal Atresia Without Fistula.** The upper pouch may vary in length as seen above, while the lower segment may be a mere atretic cord adherent to the trachea above and joined to an esophageal remnant just above the diaphragm. At the opposite extreme, there is the midesophageal stricture of Type VI.

**Tracheoesophageal Fistula Without Atresia.** The normal-sized esophagus is adherent to the lower trachea, in which area lies the fistulous opening.

**Other Abnormalities.** The presence of other abnormalities was stressed in 1919 by Plass (16) who in 94 infants, found 59 associated anomalies, 24 of which were anal atresia. In his series of 233 cases, Gross (17) in 1953 found 156 free from other defects and 77 with other defects sometimes multiple such as

Congenital heart disease	24
Malformation of anus and rectum	23
Meckel's diverticulum	10
Atresia or stenosis of small intestine	7
Coarctation of aorta	4
Malrotation of colon and intestine	4

The presence of other defects however should not exclude a surgical approach for in 45 consecutive cases Haight (18) found only 1 patient with an additional deformity incompatible with life.

## CLINICAL FEATURES

**Physical Signs.** Though few in number, these signs are definitive. All obstetricians and obstetric nurses, as well as pediatricians must fully appreciate the signs in order to make the earliest possible diagnosis.

A mother's *hydramnios* or a *premature delivery* may herald esophageal atresia. A newborn continually drooling frothy saliva or tenacious mucus or choking on his first feed has congenital esophageal atresia till proved otherwise. The mucus and saliva, easily inhaled, cause repeated attacks of choking and cyanosis which, if unrelieved, inevitably lead to atelectasis and fatal aspiration bronchopneumonia.

When a baby presents with *atelectasis* esophageal atresia is suspect. Because of the fistula from the trachea to the lower esophagus these children may occasionally show a *distended tympanitic abdomen* which increases in size with crying, but diagnosis should never be delayed till this is obvious. A thorough search especially of heart, alimentary canal, and limbs is made for associated congenital abnormalities.

*Prematurity* accompanies at least 25 per cent of the cases. Potts (19) in 1950 reported prematurity in 9 of 35 patients with esophageal atresia and Gross in 54 of 233 patients. Further prematurity increases operative mortality.

Tracheoesophageal fistula without atresia, or congenital stenosis without fistula, should be strongly suspected from a history of *cough when being fed*.

### DIAGNOSIS

Esophageal atresia is confirmed by passing a firm, small rubber catheter (size 9 English) gently down the esophagus and finding its progress arrested at 7 to 9 cm from the upper jaw. With the rarer tracheoesophageal fistula without atresia, the catheter will pass on into the stomach.

As the lesion may be detected in any hospital or nursing home and almost always some distance from surgical facilities, the use of radiopaque media to confirm diagnosis is contraindicated until the child has been transferred to the place where surgical relief will be undertaken. *On no account should barium be given*, for it usually enters and obstructs the bronchial tree.

**Mortality When Untreated.** Untreated, the lesion is usually fatal within a week, though Trump (20) reported a case surviving 37 days when given glucose intraperitoneally. Shaw and associates (21) in 1955 reported successful surgical repair on the twenty-eighth day in one child similarly kept alive.

### PREOPERATIVE MANAGEMENT

**Prevention of Complications.** The immediate problem of preventing respiratory complications, especially aspiration bronchopneumonia, requires the constant services of conscientious nurses and the closest cooperation between pediatricians and surgical staff. Arrangements must be made for immediate transfer to a thoracic surgical unit.

Meanwhile, salivary secretions are reduced by giving atropine sulfate 0.3 mg subcutaneously. To prevent aspiration of saliva, a No. 8 soft rubber catheter is passed through the nose into the blind upper pouch and aspirated either with a Dakin aspirating syringe or with electric suction.

Views vary as to whether the child should be nursed head down to promote postural drainage or sitting up to prevent regurgitation from the stomach into the trachea. Usually, however, the cardiac sphincter is competent and the child is therefore nursed on his side in an oxygen crib or Isolette and with head slightly down.

Injections of penicillin 50,000 units and streptomycin [10 mg per pound (0.5 kg)] are given 8-hourly as further prophylaxis against lung infection.

When gastric distension is great and embarrasses respiration, it may require urgent relief by preliminary gastrostomy which Shaw (21) found valuable before transporting the infant some distance for operation.

**Transfer to Hospital.** During transfer, the nurse must continue to give oxygen and aspirate the mouth and pharynx.

**Further Preoperative Management.** **RESUSCITATION OF THE PATIENT** If in poor condition, the patient is further resuscitated, and x-ray examination is delayed until immediately before operation.

**ROENTGENOGRAPHY** If reasonably fit, however, the child is brought to the x-ray department where anteroposterior and lateral films are taken. These may show an air-filled proximal pouch, a distended stomach, and, in addition, pneumonia or atelectasis, which Holt (18) reported in 27 of 45 patients, 22 being affected in the right upper lobe.

Next, with the child resting head upon the inclined x-ray table, 1 ml. of radiopaque fluid is injected down the catheter into the pouch, further anteroposterior and lateral films taken, and the fluid reaspirated. These films confirm the earlier findings and show the nature of the defect (Fig. 2).



Fig. 2. Right lateral film showing diaphragm in short proximal pouch widely separated from distal pouch which opens into trachea. This finding, plus air in stomach, shows deformity to be Swenson Type II

In two of five patients with no x ray evidence of air in the intestine, Holt found at operation, a narrow fistula showing that absence of air need not mean absence of fistula to the lower segment. Roentgenograms sometimes fail clearly to outline the side of the aortic arch, as in one of the writer's cases when right thoracotomy revealed a right sided aortic arch obstructing the approach to the lesion. The wound was closed and anastomosis done from the left side.

If there is fistula without atresia, the fistula may be demonstrated only when the child swallows watery opaque medium while lying in the prone position. This can also be combined with endoscopy (22). Again, in esophageal stenosis without fistula the swallowing of thick barium may be required to demonstrate the lesion.

**VENOCLYSIS** The patient is next taken to the ward and an intravenous drip is inserted into the umbilical vein by means of a fine polythene tube cannula. If the vein has closed, then the left long saphenous vein is used. To keep a stricter check on the amount of fluid given the standard 600 ml. infusion bottle is arranged to act as a reservoir filling a 100 ml. graduated burette and glucose and saline solution (e.g. 4.3 per cent dextrose and 0.18 per cent sodium chloride) given at the rate of 70 ml. per pound (0.5 kg.) body weight in 24 hours.

**BLOOD EXAMINATION AND TRANSFUSION** Blood from a heel stab is typed and crossmatched. From his successful experience Swenson (13) advises that within 12 hours a red blood cell count be taken, and, if below 4.5 million cells per  $\text{cm}^3$ , a small blood transfusion (not more than 60 ml.) be given. If the count is over 4.5 million plasma instead of blood should be given. Swenson found that within 18 to

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24 hours the child's temperature, if previously elevated, fell and the pulse became slower and fuller.

**CHOICE OF TIME TO OPERATE** When the diagnosis has been made early and there are no signs of respiratory infection, there should be no delay in operating. When diagnosis has been delayed, saliva has been aspirated into the lungs, and bronchopneumonia has developed, the above measures to improve the patient's condition are required, and 12 to 18 hours can be profitably spent in applying these measures. It is useless to delay operating beyond 48 hours after admission.

### THE OPERATION

After preliminary injection of atropine 0.3 mg, a general anesthesia is commenced, given by an anesthetist skilled in infant and thoracic technics. Choice depends on the anesthetist, but gas, oxygen and ether, given with a tightly fitting face mask, is satisfactory.

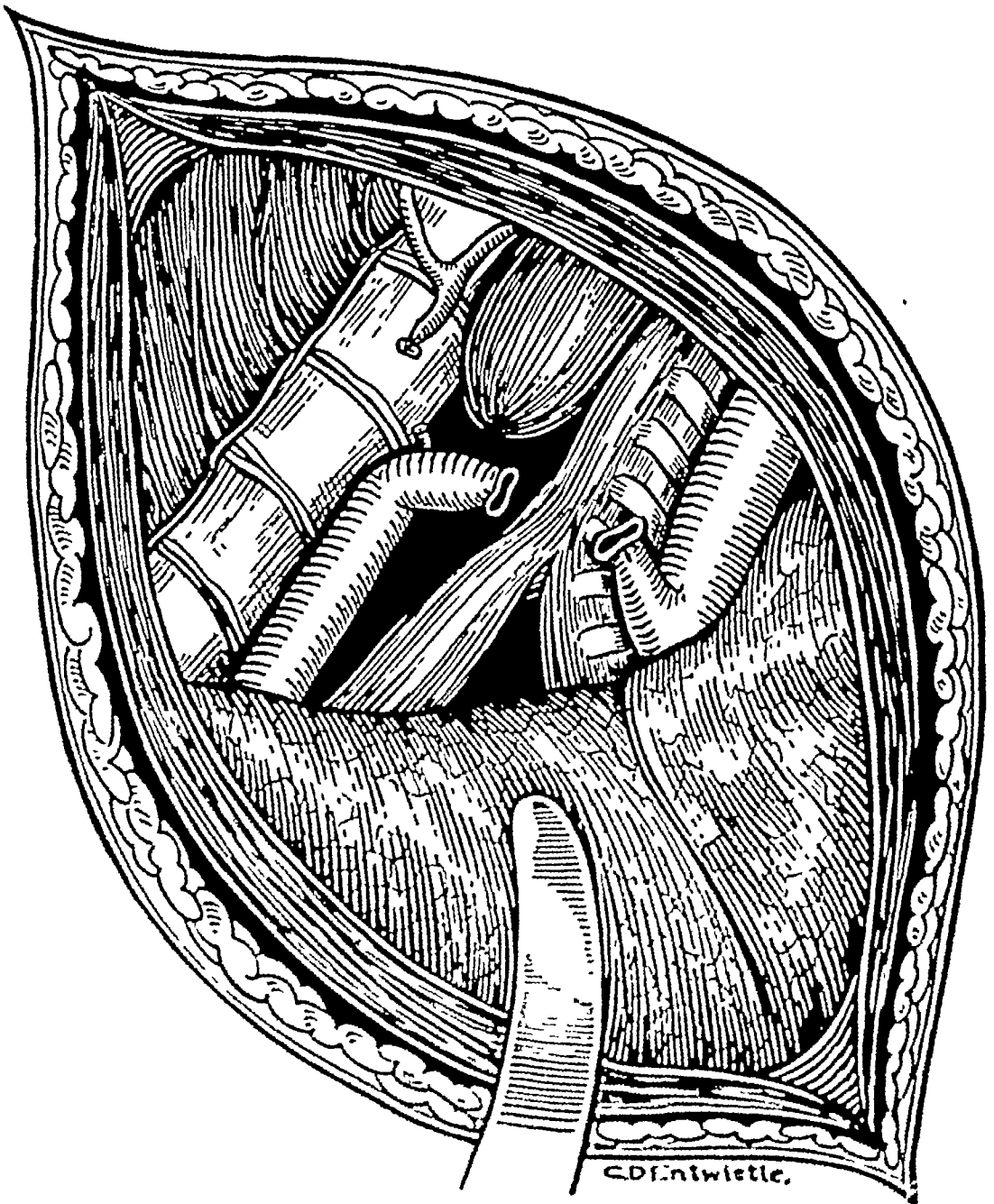


Fig. 3A Operative technique for repairing congenital esophageal atresia. Right extrapleural approach.

## The Operation

**The Approach.** While Belsey (11) has advocated a right transpleural approach, Shaw (15) prefers the extrapleural one as it minimizes anesthetic problems, gives a quieter mediastinum during dissection and suturing, and localizes any postoperative leak. The extrapleural can be converted into a pleural approach if the stomach requires mobilizing. Most recorded successes have been via the extrapleural route.

When roentgenograms show a right-sided aorta, a left thoracotomy is required.

**Principles of the Operation (Fig. 3A)** With the infant on his left side and the right arm forward, a 4 to 5 cm incision is made parallel to the vertebral border of the scapula, and the fourth rib is exposed by muscle retraction in the auscultatory triangle and resected. By careful blunt dissection, the extrapleural plane is developed and a rib spreader inserted. After doubly ligating and dividing the azygos venous arch the mediastinum is entered.

The *upper segment* more easily identified if the anesthetist gently pushes a catheter into it, is dissected from the trachea and mobilized high into the neck. As its good blood supply and hypertrophied wall allow of lengthening to bridge any gap it is sutured to the fibrous prevertebral fascia as low as possible as suggested by Swenson.

The *lower segment* is encircled by a fine tape or ligature, gently dissected as high as possible and divided close to the trachea. The tracheal fistula is closed with interrupted 5-0 silk sutures on atraumatic needles.

**The Anastomosis (Fig. 3B)** The success of the operation depends on the anastomosis. Great care is therefore essential and the segments should be delicately handled with forceps. Because the lower segment receives some blood supply from the trachea and because after division, its narrow upper end may become cyanotic, Belsey (11) advises resecting this portion. Shaw (15) however, advises first inserting a posterior layer of fine silk sutures from the upper segment to the desired level on the lower segment, tying these, and then trimming the lower segment back. The mucosa of the upper segment is next opened, mucosal anastomosis completed, and the final layer of sutures inserted.

The chest wall is closed with a small water seal drain into the extrapleural space which is brought through a separate paravertebral stab incision. When a transpleural approach has been used, the mediastinal pleura is left unsutured, but the chest is closed over a water seal drain. Lung re-expansion is checked by a chest film.

Blood transfusion up to 200 ml of blood to replace blood loss is given during the operation. If the intravenous cannula becomes blocked during that time the writer has still replaced blood by injection into the superior vena cava with a fine needle and a 20 ml. syringe.

After operation, aspiration bronchoscopy is advisable thereafter a Stamm gastrostomy is performed. (See under Feeding, p 247.)

**Inadequate Lower Segment.** When exploration shows partial or complete atresia of the lower segment or too large a gap for primary anastomosis, the lesion can still be treated at that time from the right side. Byron (23) Madden (24), and others have enlarged the esophageal hiatus reflected peritoneum from off the stomach, divided the left gastric artery and performed immediate transthoracic esophagogastric anastomosis. Gross (17) advises sliding the stomach up through a dilated esophageal hiatus to permit anastomosis of the two esophageal segments.

If, however the child is in poor condition, then the fistula is divided and sutured, the chest wound closed, and left cervical esophagostomy and gastrostomy performed. Eighteen months later the esophagus is reconstructed by Sweet's (25) method.



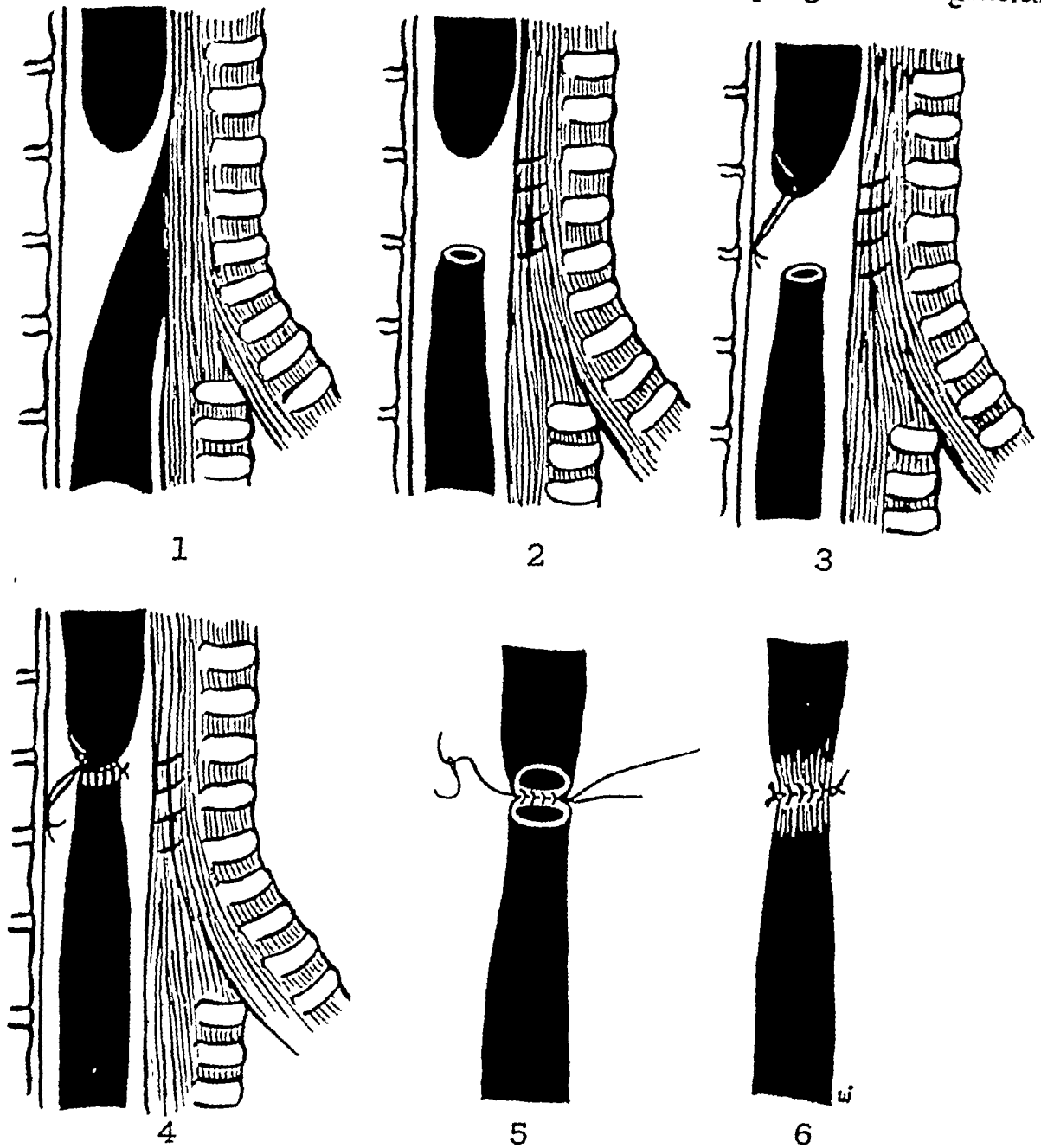


Fig 3B Stages in dissection and anastomosis 1, The deformity to be corrected 2, The distal esophagus has been separated from the trachea and the fistula closed 3, The dissected proximal esophageal pouch is sewn down to the prevertebral fascia 4, The anastomosis completed 5 and 6, The anastomosis, preferably with interrupted 5-0 silk sutures on curved atraumatic needles

Through a left thoracoabdominal approach, the gastrostomy is closed by interrupted sutures in the long axis of the stomach, and the stomach is mobilized and drawn through the left chest to the first left intercostal space and tethered. After the chest wound is closed, the patient is turned on his back and the inner half of the left clavicle, first rib and costal cartilage resected, and the stomach anastomosed to the upper pouch. An alternative method advocated by Shaw and Paulson (15) is intrathoracic, substernal placing of a Roux-Y jejunal loop.

#### POSTOPERATIVE MANAGEMENT

**Prevention of Respiratory Complications.** The patient is returned to a warm oxygen crib, under the care of a special nurse. The pulse and respiration are recorded half-hourly. All preoperative nursing measures are continued, including antibiotics.

nursing in Trendelenburg position with two-hourly turning from side to side and frequent aspiration of the nasopharynx. This further stimulates a cough reflex. As nasal or esophageal tubes left in position would interfere with normal swallowing reflexes, irritate mucous membranes, and increase the chances of pulmonary infection, they are not reinserted after the operation.

Check roentgenograms are taken the day after operation, then daily for five days and thereafter as required. The mediastinal drain should remain in for four to five days as a safeguard against a leaking anastomosis. If aspiration bronchoscopy at the end of the operation shows pneumonia or excess tracheal secretions or if there is edema of the glottis that does not respond to one further bronchoscopy then tracheotomy is required.

**Feeding.** Because of the real danger of fluid overload (13-15) it is not advisable to persist with the drip infusion after the operation. Details of an accepted regime are listed by Roberts and associates (28).

Oral feedings with or without a tube are withheld until the anastomosis is well healed. Stamm gastrostomy is required, and is performed either at the close of the operation (17) or 24 hours later under local anesthesia (13).

Eight hours later half ounce (15 ml.) feedings of sterile water or oral electrolyte solution (26) are given slowly via the gastrostomy tube and are continued every two hours, increasing to 1 oz. (30 ml.) per feeding in 24 hours. Thereafter a diluted milk regime is substituted and increased to normal strength within six days.

On the fourteenth day if convalescence has been smooth, four hourly oral feedings of sterile water are commenced and gradually increased. As there is often some degree of stricture formation, oral feeding should not be forced. The gastrostomy tube is left in site for six weeks as a further safeguard and is not removed until after a check esophagram to determine if a fistula has formed.

### ANASTOMOTIC COMPLICATIONS

**Leaking Anastomosis.** Though serious this is not inevitably fatal, especially if an extrapleural approach has been used. The fistula develops through the drainage wound and in time heals by continued drainage of the mediastinum. If it has not already been done gastrostomy is required. When the approach has been transpleural only reanastomosis using, as Belsey (11) suggests, fine stainless steel wire can hope to save the day.

**Tracheal Fistula.** A recurrent tracheoesophageal fistula due to leaking anastomosis requires immediate reoperation.

Shaw and associates (21) reported nine recoveries from leaking anastomosis and five from tracheal fistula.

**Stenosis.** Although following repair many infants have some esophageal stricture it seriously affects only a few (Fig. 4). It usually responds to gentle esophagoscopic dilatation under general anesthesia. When the stricture is resistant to weekly dilatations reoperation and excision of the stricture are required. Gross (17) reported that 34 of the 97 recoveries from endothoracic anastomosis required dilatation and that it was most troublesome during the first year of life lessening thereafter



Fig 4 Postoperative esophagram after successful surgical treatment. Stenosis was treated with dilatations at fortnightly intervals.

### RESULTS OF SURGICAL TREATMENT

Early reports of surgical treatment were encouraging. By 1946, Haight (12) had 9 children alive from 16 operations, while in 1947 Swenson (13) reported successes in 15 patients, an operation mortality of 6.6 per cent.

In 1933, Gross (17) reported on 233 cases, of which 224 had surgical treatment with 109 survivals. Of these patients, 12 had multiple-stage construction of an atresia thoracic esophagus, 97 had endothoracic esophageal anastomosis, which included 5 in whom the stomach had been brought into the chest. The survival rate in operations during 1953 was 67 per cent.

More recently, reports from Humphreys and his associates (25) and Shaw (14) support the view that a salvage rate of 66 per cent can be achieved, but that delay in diagnosis, poor condition when referred for surgery, and associated anomalies incompatible with life make improvement of these results difficult to achieve.

### CONCLUSION

Awareness of esophageal atresia, prompt recognition, and surgical treatment before respiratory complications can occur offer the best hope of success. In skillful hands, at present two thirds of all cases seen can be expected to survive operative treatment.

### REFERENCES

## References

- 4 Keith, A. A demonstration on constriction and occlusions of the alimentary tract of congenital or obscure origin, *Brit. M J* 1 301 1910
- 5 Ladd, W. E. The surgical treatment of esophageal atresia and tracheo-esophageal fistulas, *New England J Med.* 230 625 1939
- 6 Leven, N. L. Congenital atresia of esophagus with tracheo-esophageal fistula. Successful extrapleural ligation of fistulous communication and cervical esophagostomy *J Thoracic Surg.*, 10 648 1941
- 7 Lanman, T. H. Congenital atresia of the esophagus 32 cases, *Arch. Surg.*, 41 1060 1940
- 8 Shaw R. R. Surgical correction of congenital atresia of the esophagus with tracheo-esophageal fistula, *J Thoracic Surg.*, 9 213 1939
- 9 Haight, C., and Towaley H. A. Congenital atresia of esophagus. Extrapleural ligation of fistula and end-to-end anastomosis of esophageal segments, *Surg. Gynec., & Obst.*, 76 672 1943
- 10 Franklin, R. H. Congenital atresia of the esophagus. Two cases successfully treated by anastomosis, *Lancet*, 2 243 1947
- 11 Belsey R. H. R., and Donnlson, C. P. Congenital atresia of the esophagus, *Brit. M J.*, 2 324 1950.
- 12 Vogt, E. C. Congenital esophageal atresia, *Am. J Roentgenol.*, 22 463 1929
- 13 Swenson, O. The diagnosis and treatment of atresia of the esophagus and tracheo-esophageal fistula, *Pediatrics*, 1 195 1948
- 14 Borrie, J., in R. H. Maingot's *The Management of Abdominal Operations*, 2nd ed., London, H. K. Lewis & Co., Ltd., 1957 Vol. I, p 478
- 15 Shaw R. R., and Paulson, D. L. Congenital anomalies of the esophagus, *Am. J Surg.*, 93 196 1957
- 16 Plass, E. D. Congenital atresia of the esophagus with tracheo-esophageal fistula, *Johns Hopkins Hosp Rep.*, 18 259 1919
- 17 Gross, R. E. *The Surgery of Infancy and Childhood*, Philadelphia, W. B. Saunders Co., 1953
- 18 Holt, J. F. Haight, C., and Hodges, F. J. Congenital atresia of the esophagus and tracheo-esophageal fistula, *Radiology* 47 457 1946
- 19 Potis, W. J. Congenital atresia of the esophagus with tracheo-esophageal fistula, *J Thoracic Surg.*, 20 671 1950
- 20 Trump F. A. Congenital esophagotracheal fistula Report of a case living thirty seven days on glucose intraperitoneally *J Pediat.* 2 212, 1933
- 21 Shaw R. R., Paulson, D. L., and Siebel, E. K. Congenital atresia of esophagus with tracheo-esophageal fistula. Treatment of surgical complications, *Ann. Surg.*, 142 204 1955
- 22 Herweg, J. C., and Ogura, J. H. Congenital tracheo-esophageal fistula without esophageal atresia *J Pediat.*, 47 293 1955
- 23 Byron F. X. Congenital atresia of the esophagus with hypoplasia or agenesis of the lower segment, *Surgery* 24 841 1948.
- 24 Madden, J. L. Congenital atresia of the esophagus treated by one stage primary esophago-gastrostomy employing a right transpleural approach, *J Thoracic Surg.*, 21 460 1951
- 25 Sweet, R. H. A new method of restoring continuity of the alimentary canal in cases of congenital atresia of the esophagus with tracheo-esophageal fistula not treated by immediate primary anastomosis, *Ann Surg.* 127 757 1948
- 26 Flett, J., Pratt, E. L., and Darrow D. C. Methods used in treatment of diarrhea with potassium and sodium salts, *Pediatrics*, 4 604 1949
- 27 Humphreys, G. H. Hogg, B. M., and Ferrer J. Congenital atresia of esophagus, *J Thoracic Surg.* 32 332, 1956.
- 28 Roberts, K. D. Carré I. J., and Inglis, J. McN. The management of congenital oesophageal atresia and tracheo-oesophageal fistula, *Thorax*, 10 45 1955

## BLEEDING FROM ESOPHAGEAL VARICES

Bleeding from esophageal varices, be it minor or massive, single or repeated, constitutes a perplexing problem in the surgical management of portal hypertension. It may occur as the first sign of the disease, or after many and varied operations.

**Historical Note.** In 1945, following the papers of Whipple (1) and Blakemore (2, 3), there was renewed interest in the treatment of portal hypertension and Banti's Syndrome. Since then, many operations have been evolved, both direct and indirect, which are summarized by Cooley and DeBakey (4). For the nonurgent, uncomplicated case, end-to-side portacaval anastomosis through a right thoracoabdominal incision remains the most rational and satisfactory procedure (5); many patients so treated have been relieved of their previous symptoms. Nevertheless, there remains a substantial group of portal hypertensive patients, with or without previous shunt operations, in whom the *hemorrhage may be so sudden and so severe* as to constitute a serious threat to life.

It is to the management of this latter group that this chapter refers.

### PATHOLOGY

In order to appreciate the problems of massive bleeding from esophageal varices, it is important to understand the process that causes portal hypertension.

**Causes of Portal Hypertension.** Hypertension is present when the pressure in the portal vein—with the recording manometer and vein at zero level—measures over 150 mm. of water (6). The obstruction causing portal hypertension may be *prehepatic*, *hepatic*, or *posthepatic*, and, of these three, the first two are of practical importance.

**PREHEPATIC OBSTRUCTION** In this condition, either the main portal vein or its intrahepatic branches are obstructed by one of three abnormalities. There may be *simple stenosis* of the portal vein, *atresia* for a variable length, or *cavernomatous transformation* in which the vein is replaced by a mass of varicose channels. The characteristic feature of prehepatic obstruction is that the liver is macroscopically and microscopically normal. Milnes Walker (5) recorded portal pressures increased to as much as 300 to 450 mm. of water, with an average of 386 mm. *This hypertension causes abnormal venous connection, especially esophageal varices, which are responsible for the one symptom, namely, hematemesis.* Symptoms in prehepatic obstruction usually arise early in life.

**INTRAHEPATIC OBSTRUCTION** This is the result of intrahepatic fibrosis which *macroscopically* may show a firm liver almost normal in size with raised areas up to 2 mm. in diameter on its surface. Alternatively, the liver is shrunken and covered with nodules up to 2 cm. in diameter.

*Microscopically* three characteristic types occur with

- 1 Increased fibrous tissue only in the portal tracts, but with normal parenchyma and central veins;

2. Fibrous tissue spreading more diffusely through the parenchyma toward the central veins
3. Liver structure completely disorganized and with bile ducts, arteries, and veins compressed in masses of fibrous tissue

**Relation of Symptoms to Lesions.** In type 1 with portal-tract fibrosis, the liver cells and liver function tests are normal but portal venous pressures are high. Type 2 with diffuse hepatic fibrosis, follows type 1 with no serious impairment of hepatic function and with high portal vein pressures. Type 3 with widespread postnecrotic scarring compressing all elements of the liver shows the clinical picture of impaired liver function added to portal hypertension.

### CLINICAL FEATURES

This disease affects all age groups. When presenting with *hematemesis* and *melena* these patients are usually first seen by physicians and may be referred for surgical opinion shortly after the first episode or because of recurring hemorrhages. When the *hematemesis* is *uncontrollable* however they are usually referred earlier but not before considerable bleeding has occurred and vigorous transfusion has been necessary.

Physical examination in the young usually reveals little abnormality at first. Later as the disease progresses the spleen may become palpable. In adults, especially if the condition is long standing and associated with portal cirrhosis there may be ascites, a small cirrhotic liver, caput medusae, and hemorrhoids.

**Investigations.** Patients with severe *hematemesis* fall into two groups:

1. Those where previous investigations have established the diagnosis and
2. Those presenting for the first time. This latter group may be seen either with bleeding temporarily arrested or with it continuing unabated.

When bleeding has been arrested, there are four important investigations designed to establish the diagnosis, to determine the state of the liver and the degree of portal hypertension, and to aid in making the best choice of operation. They are:

1. Blood examination
2. Fluoroscopic examination of the esophagus, stomach, and duodenum
3. Liver function studies
4. Esophagoscopy

**BLOOD EXAMINATION.** Red-cell and hemoglobin estimations determine the degree of anemia. Serum albumin levels are a guide to liver function (see later), and a Wassermann test excludes a luetic cause.

**FLUOROSCOPY.** Esophagrams aim at excluding other causes of *hematemesis* such as reflux esophagitis, gastric and duodenal ulcers, or neoplasm. In portal hypertension the characteristic soap-bubble appearance of the lower esophagus is usually seen (Fig. 1 left) but as varices are not always demonstrated by this method, a negative result does not exclude their presence.

Regarding *portal venography* as the aim is to demonstrate patency of the portal veins and their suitability for portosystemic anastomosis, this investigation is beyond the field of this article. However portal venography should always be performed when the patient has recovered from his hemorrhage and as a prelude to a planned anastomosis. It was originally carried out under direct vision during operation but



Fig 1 Esophagram showing soap-bubble effect typical of esophageal varices. Right, Esophagram after transection of cardia, ligation of varices and re-anastomosis, showing no evidence of varices.

has more recently been advocated before operation. The technic involves percutaneous injection of a radiopaque medium, such as diiodone, into the spleen. The medium rapidly enters the venous system and is carried to the liver in sufficient concentration to show the course of the veins. Walker reported 12 such venograms with satisfactory results (8).

**LIVER FUNCTION TESTS (9)** Though patients with normally functioning liver may have portal hypertension without ascites, when the liver is damaged not only is there a greater chance of ascites but the ultimate prognosis is poor. Linton (9) warns that, while clinical assessment of necessity takes priority over tests, the most serious risk is a patient with a plasma albumin below 3 gm per 100 ml with ascites that fails to respond to medical therapy and with a 3+ or 4+ cephalin flocculation and a prothrombin time that does not respond to vitamin K therapy. On the other hand, a good-risk patient has a 1+ to 2+ cephalin flocculation, a plasma albumin greater than 3 gm per 100 ml, absence of ascites, a bromsulfalein-retention test below 10 per cent after one-half hour, and a prothrombin time within four seconds of normal.

**ESOPHAGOSCOPY.** This is the only satisfactory way of confirming the presence of the esophageal varices (10). They are clearly seen bulging through the mucosa into the esophageal lumen, can be of enormous size, and, though occurring throughout the greater length of the esophagus, are most obvious at the cardia.

When bleeding has been arrested, the pressure within the varices as a guide to pressure in the portal system is measured by Allison's (11) method, using a long

needle mounted on an endoscopic suction tube and attached to an electromanometer. The method is of great value both before and after portasystemic anastomosis in estimating the efficacy of the shunt.

**Prognosis.** Bleeding constitutes the gravest hazard to patients with esophageal varices. Linton (7) reported an alarmingly high incidence of deaths from hemorrhage in patients with cirrhosis of the liver and Banti's syndrome who were treated purely along medical lines.

Nachlas and associates (12) found that in 62 patients proved to have only varices as their source of hemorrhage 66 per cent died from their first hemorrhage. A patient who survived this episode was calculated to have two chances out of three of being alive at the end of one year.

Eppinger estimated that about 20 per cent of patients with cirrhosis of the liver die of hemorrhage before ascites or hepatic insufficiency supervene (quoted by Cooley 4).

With such poor prospects from medical treatment, any emergency surgical measure that will effectively arrest the hemorrhage and render the patients safe and fit for definitive treatment of his portal hypertension is surely warranted.

### MANAGEMENT

There are *three principles* in the logical treatment of these seriously bleeding patients. They are

- 1 *Temporary arrest* of the bleeding esophageal varices by tamponade of the cardioesophageal region with an intragastric balloon, followed by
- 2 *More permanent control* of the bleeding by direct operation on the varices. Once the hemorrhagic episode has passed,
- 3 *Definitive treatment* is undertaken to lower portal venous pressure by venovenous anastomosis, producing either an end-to-side portacaval shunt or an end-to-side splenorenal shunt.

**Temporary Arrest (Cardioesophageal Tamponade)** This method, first used by Rowntree and associates (13), was modified by Sengstaken (14) who devised a twin balloon with triple lumen tubing, of which one was for feeding purposes and the other two were for inflating the balloons. As Linton (7) points out, however the upper balloon is unnecessary because the bleeding is actually controlled when traction on the tube causes the lower balloon to compress the submucous veins at the cardia, thereby preventing portal blood from entering the varices (Fig 2).

The importance of obstructing the flow of blood from stomach to esophagus cannot be overemphasized. Even though the *actual bleeding point* on the varix be 4 to 5 cm. above the cardia and therefore not directly occluded by the balloon, nevertheless, the hemorrhage ceases.

**TECHNIC** An adequate balloon can be made by carefully sealing Penrose rubber tubing on to a Levin tube. After testing, the tubing is inserted through the mouth, inflated to approximately two inches in diameter and a two-pound traction weight applied. Roentgenograms as in Figure 2 help with the correct positioning of the tube. The method however has severe limitations for as pressure by the tube or balloon may ulcerate the pharynx or cardioesophageal junction it requires removal in 8 to 12 hours. Further when the balloon is deflated and withdrawn, bleeding frequently starts again and can prove fatal.





Fig 2 Sengstaken tube has been passed into stomach of patient having massive hemorrhage. Lower balloon is inflated prior to fixing weight traction.

More recently, Kehne (15) has reported from laboratory and clinical observations that 10 to 20 units of surgical pituitrin injected in the arm at the rate of 2 units per minute have effectively lowered portal venous pressure. They advocate its use as an adjunct to tamponade in the emergency treatment of esophageal bleeding.

Because of the limitations of tamponade, therefore, these patients require more effective arrest of their bleeding, not only as a lifesaving measure but also as a means of tiding them over until fit enough for definitive shunting of the portal system by venovenous anastomosis.

*Three different principles have been used:*

- 1 Direct suture of the varices via esophagotomy,
- 2 Division of the junction between the portal system and the esophageal veins by transection and resuture of the cardia,
- 3 Excision of the varix-bearing portion of the esophagus by (a) partial esophagogastrectomy or (b) subtotal esophagectomy.

**Direct Suture of the Esophageal Varices.** In 1949, Boerema (16) performed multiple intraesophageal ligation of the varices and obliterated them between ligatures by injecting sclerosing solution. In 1950, Crile (17) reported performing intraesophageal ligation of varices in seven patients who had bled after splenectomy. The operation, however, was a *planned procedure* and was not done at the time of bleeding. Others have reported parallel results. In 1953, however, Linton reported success with 14 *emergency operations*.

**TECHNIC** Linton, using Crile's method, first inserts a balloon tube to control bleeding by cardioesophageal tamponade. With the balloon tube still in place and

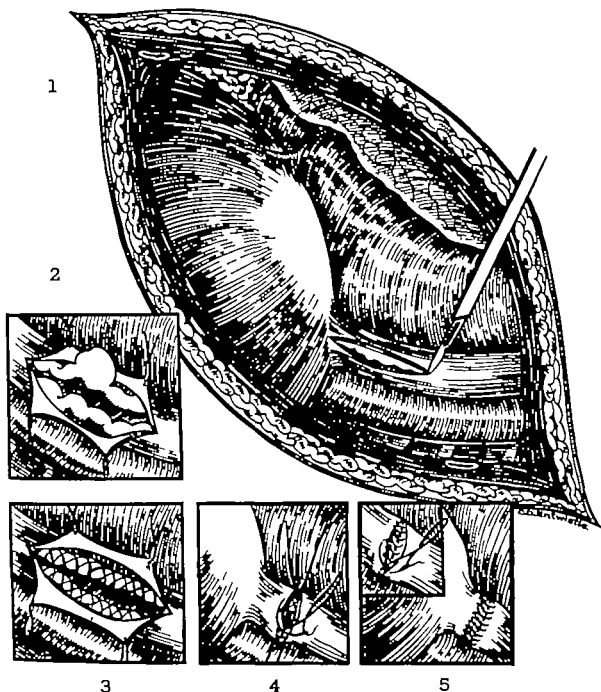


Fig. 3 Technic of transesophageal suture-occlusion of varices. 1 The esophagus is exposed in a left thoracotomy and vertically incised. 2, The group of submucosal varices are easily identified. 3 They are obliterated with continuous catgut sutures. 4 and 5 The esophageal incision is closed transversely with three rows of interrupted fine silk sutures.

the patient anesthetized with a cuffed endotracheal tube and lying on his right side the left chest is opened through the bed of the resected left seventh rib. The esophageal hiatus is opened to aid the approach to the stomach. An incision 5 to 6 cm long is made in the esophagus extending down to the cardia of stomach (Fig. 3).

The bleeding varices and other varices are picked up leaving the mucosa intact. Using an intestinal atraumatic needle with 0 chromic catgut, each column of varices is obliterated with a continuous suture from the stomach below the esophagus for 6 cm. In practice three such columns are isolated and sutured.

After completing hemostasis within the esophagus and aspirating all old blood from the stomach lumen, the esophageal incision is closed transversely with three rows of interrupted fine silk sutures.

The usual postoperative measures for thoracotomy apply, and a fluid diet is given for the first 10 postoperative days.

Of the 14 patients reported by Linton, only 1 died. As bleeding recurred at intervals varying from two weeks to five months after operation in five patients so treated, it is clear that a portacaval shunt of some type should be carried out as soon as the patient's condition warrants it.

Regarding *definitive treatment* of the portal hypertension in the remaining 13 patients, 3 with Banti's syndrome and previous splenectomy could have no shunt because of cavernomatous changes surrounding the portal vein. In another, the operation was performed after both splenorenal and portacaval shunts had failed to prevent further bleeding. In eight others, shunts had been planned or performed.

**Transection of the Cardia.** This procedure, a further development of Tanner's *gastric transection* (19), aims at cutting off the portal blood flowing to the esophageal varices. The lower esophagus is exposed through the left pleural cavity, clamped, divided, and resutured in two layers after ligating the larger submucous varices. A barrier of scar tissue thus forms, and there is time for more adequate collateral circulation to develop elsewhere.

The technic is illustrated in Figure 4. Milnes Walker (1952) reported satisfactory results on six patients; in only one was there further bleeding in a two-year period.

**CASE REPORT.** Mrs. P. W., aged 33 years, was referred by her physician on February 26, 1953, with a two-day history of profound hematemesis and melena from esophageal varices. In 1949 she had had hematemesis, and again in 1952, when two operations were performed, namely (a) transthoracic ligation of peri-esophageal veins on March 28, 1952, and (b) transabdominal splenectomy on May 16, 1952. Inspection at that time had revealed splenic-vein thrombosis. Because of cholelithiasis, she had had cholecystectomy on September 17, 1952, when liver biopsy showed no hepatic fibrosis (Fig. 1, left).

On March 26, 1953, transection of the cardia was performed. Because of the patient's past history and the unabated bleeding, direct approach to the varices was advised. Because of previous favorable experience in nonurgent cases, it seemed reasonable to transect and resuture the esophagus in this acute emergency.

Through a left thoracotomy, pulmonary adhesions were divided and the esophagus isolated (Fig. 4A). An inspection incision was made in the diaphragm but was closed again because of dense perigastric adhesions. The esophagus was completely freed in its lower third and the gastric fundus drawn up through the esophageal hiatus into the chest. Transection and resuture were successfully performed (Fig. 4B). Three liters of blood and 1.5 liters of dextran were given during the operation.

Postoperative roentgenograms showed disappearance of the varices (Fig. 1, right). The patient remained well until April 22, 1954, when a further, smaller hematemesis occasioned a gastric transection and resuture across the incisura angularis.

On March 16, 1956, esophagoscopy confirmed that the varices had disappeared. Gastroscopy showed that the gastric mucosa proximal to the line of gastric transection was normal in color but atrophic and without rugae or any prominent veins.

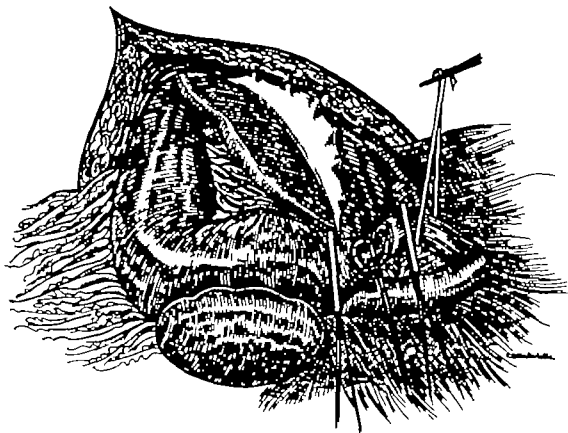


Fig. 4A. Technic of transection of cardia in a left thoracotomy. Isolation and transection of esophagus.

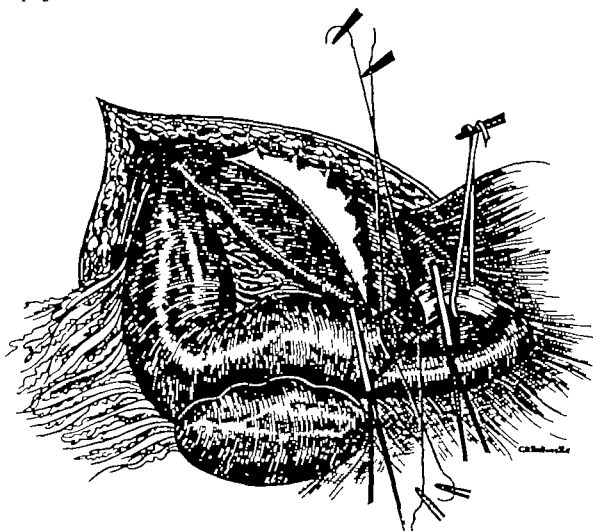


Fig. 4B Re-anastomosis with two layers of interrupted silk sutures.

**Esophageal Resection.** Methods of esophageal resection aim at removing the varix-bearing esophagus and establishing continuity by anastomosing the stomach to the esophageal stump

In 1947, Phemister and Humphreys (20) reported success with gastroesophageal resection for bleeding varices. In 1954, Cooley and DeBakey (5) advocated a more extensive resection. After an exploratory laparotomy at which portal vein pressures are measured and liver biopsy taken, the stomach is mobilized and splenectomy and Fredet-Ramstedt pyloromyotomy performed. Thereafter, the chest is entered through the fourth right intercostal space, the esophagus freed and resected from the stomach below, and a high anastomosis made above the level of the azygos arch between the stomach and the upper third of the esophagus. Cooley reported two successes in three operations.

Gastroesophageal resection undoubtedly can control severe hematemesis from varices, but personal experience has confirmed that it is a long operation, lasting four or even five hours, and technically complicated by abnormal venous channels in patients with poor liver function. As the immediate aim is to arrest hemorrhage, and as this can be done as effectively and more safely by direct ligation of the varices, or transection and resuture at the cardia, these shorter procedures are advised for this *emergency*.

There is, however, a real place for resection of the varix-bearing lower esophagus as a *planned operation* in patients with repeated bleeding after previous splenectomy and unsuccessful shunt operations. The most suitable operation then is probably esophagogastrectomy with esophagojejunostomy, using the Roux Y technic (21, 22).

Nachlas (23) supports the view that removal of the lower end of the esophagus is the most logical approach if it can be accomplished easily, quickly, and safely. In 1956, following Berman's (24) use of plastic tubes for palliating an esophageal carcinoma, Nachlas advocated resecting the lower esophagus and reestablishing esophagogastric continuity by means of a polyethylene tube. Of 5 patients subjected to this procedure, the first 2 died during the early postoperative period, and the other 3 were alive after 13, 10, and 9 months, respectively. This work is still under review.

## CONCLUSIONS

When there is massive hematemesis from esophageal varices and the patient has required two-liters or more of transfused blood without cessation of the bleeding, it is useless to temporize. Transient control, allowing sufficient time to replace blood loss, is obtained by esophageal tamponade. Thereafter the simplest effective operation to arrest the bleeding and not unduly endanger the life of a patient already debilitated should be done. The choice lies between transesophageal ligation of the varices and transection of the cardia, as the latter ensures that all varices are interrupted, it is to be favored.

At no time should splenectomy be done alone, for at a later date venovenous anastomosis will be required as a direct means of lowering the portal hypertension. In Banti's syndrome, the splenic vein is the only one available for such a shunt. When both shunts have been performed and the patient still bleeds, resection of the varices is the operation of choice.

## REFERENCES

- 1 Whipple, A. D The problem of portal hypertension in relation to the hepato-splenopathies *Ann. Surg.*, 122 449 1945
- 2 Blakemore, A. H and Lord, J W The technic of using vitallium tubes in establishing portacaval shunts for portal hypertension *Ann Surg.*, 122 476 1945
- 3 ——— Portacaval shunting for portal hypertension *Surg., Gynec. & Obst.* 94 443 1952.
- 4 Cooley D A., and DeBailey M E. Subtotal esophagectomy for bleeding esophageal varices, *Arch. Surg.*, 68 854 1954
- 5 Walker R. M Portacaval anastomosis *Lancet* 1 57 1957
- 6 ——— The pathology and treatment of portal hypertension, *Lancet* 1 729 1952.
- 7 Linton, R. R. The emergency and definitive treatment of bleeding esophageal varices *Gastroenterology* 24 1 1953
- 8 Walker R. M Middlemiss, J H and Nanson, E. M Portal venography by intrasplenic injection, *Brit. J Surg.*, 40 392, 1952
- 9 Linton, R. R. The selection of patients for portacaval shunts with a summary of the results of 61 cases, *Ann. Surg.* 134 433 1951
- 10 Carter M. G., and Zarncheck, N Esophagoscopy in upper gastrointestinal bleeding, *New England J Med.* 242 280 1950
- 11 Allison, P The measurement of blood pressure in esophageal varices, *Thorax*, 6 325 1951
- 12 Nachlas, M M., O'Neil J E., and Campbell, A J A. The life history of patients with cirrhosis of the liver and bleeding esophageal varices, *Ann. Surg.*, 141 10 1955
- 13 Rowntree, L. G., Zimmerman, E. F., Todd, M H and Ajac J Intra-esophageal venous tamponade: its use in a case of varical hemorrhage from the esophagus, *J.A.M.A.* 135 630 1947
- 14 Sengstaken, R. W and Blakemore, A. H. Balloon tamponade for control of hemorrhage from esophageal varices, *Ann. Surg.* 131 781 1950
- 15 Kehne, J H., Hughes, F A. and Gompertz, M L. The use of surgical pituitrin in the control of esophageal varix bleeding; an experimental study and report of two cases, *Surgery* 39 917 1956.
- 16 Boerema, I. Bleeding varices of the esophagus in cirrhosis of the liver and Banti's Syndrome, *Arch. Chir. Neerl* 1 253 1949
- 17 Crile, S Transesophageal ligation of bleeding esophageal varices a preliminary report of 7 cases, *Arch. Surg.*, 61 654 1950
- 18 Hallenbeck, G A., and Shockett, E. Treatment of bleeding esophageal varices after splenectomy *Arch. Surg.*, 71 581 1955
- 19 Tanner N C Discussion Gastroduodenal hemorrhage as a surgical emergency *Proc. Roy Soc.*, 43 147 1950
- 20 Phemlister D B., and Humphreys, E. M Gastro-esophageal resection and total gastrectomy in the treatment of bleeding varicose veins in Banti's Syndrome *Ann. Surg.* 126 397 1947
- 21 Allison, P R. and da Silva, L T The Roux loop *Brit. J Surg.*, 41 173 1953
- 22 Borne, J in *Malngot's Management of Abdominal Operations*, 2nd ed., London, H. K. Lewis & Co. 1957 Vol I, p 459
- 23 Nachlas M M Treatment of bleeding esophageal varices by resection of the lower esophagus, re-establishment of esophagogastric continuity by means of a polyethylene tube, *Arch. Surg.* 72 634 1956
- 24 Berman, E. F The experimental replacement of portions of the esophagus by a plastic tube *Ann. Surg.*, 135,337 1952.

## CORROSIVE BURNS OF THE ESOPHAGUS

As the proper emergency handling of these unfortunate patients can have such far-reaching effects, all medical practitioners should be fully aware of the problems involved. The key to successful management lies in clearly appreciating the progress of the pathology from burn to stricture formation.

### PATHOLOGY

Although in some adults the corrosive is taken with suicidal intent, accidental swallowing of lye (caustic soda) by children accounts for most of the cases seen. Analyzing 233 cases of chemical esophageal burns, Carver and associates (1) found that most were due to drinking lye and that 188 occurred in children less than 6 years old.

The severity of the burn depends on the substance swallowed, its chemical nature, concentration, and quantity, and on the duration of contact with the esophageal mucosa. In addition to lye, other corrosives such as ammonia, lysol, iodine, and sulfuric and nitric acids have all been encountered. The speed of destruction by lye was experimentally determined by Krey (2) in rabbits. He found that a normal NaOH solution in contact with esophageal mucosa for 10 seconds necrosed through mucosa and submucosa to involve some of the inner longitudinal muscle, 3N necrosed to the circular muscle, 5N to the outer longitudinal muscle, and 7N penetrated to the esophageal wall.

*Pathologically as well as clinically, esophageal burns fall into three groups:*

- 1 Those that are immediately fatal from the general as well as the local effects of the corrosive,
- 2 Those that are mild and produce a reversible esophagitis;
- 3 Those that produce a severe esophagitis.

It is this last group with which this chapter is mainly concerned.

Regarding *site*, the severest burns—leading to the tightest strictures—tend to occur in the middle and lower thirds of the esophagus.

The chain of events, from burn to established stricture, occupies approximately a two-month period which can be divided into three distinct phases:

- 1 *The Acute Phase of Necrosis* This lasts up to 10 days; it commences with mucosal necrosis and is followed by sloughing and associated inflammatory reaction of all esophageal layers.
2. *The Intermediate Phase of Ulceration* This is ushered in with the separation of the mucosal slough which passes down the alimentary canal leaving behind an infected, granulating, tubular ulcer the length of the esophagus. This phase may last no more than seven days or as long as four weeks. Certainly, as Krey has shown, 15 days after injury actual stricture formation begins as the collagen fibers commence to contract.

- 3 *The Final Phase of Stricture Formation* The ulcer is gradually replaced by fibrous tissue which steadily contracts to a tight *stricture*. This is usually complete within two months but may take up to a year. Of practical importance is that the severity of this final phase of stricture formation is closely related to the adequacy of early treatment.

The foregoing train of pathologic events was also observed in experimental lye burns of the esophagus by Boshier and associates (3).

**Complications.** These are many and varied. They include the immediate complications of starvation and acute dehydration *during the time of the burn*, penetration through the esophagus into the mediastinum or great vessels with fatal hemorrhage *during the phase of ulceration*, food impaction, or esophageal perforation and mediastinitis from the use of dilating bougies *in stricture formation* and sometimes there is the final remote complication, years later of neoplastic formation.

### CLINICAL FEATURES

**Serious Burns.** *In the acute phase* with burns and blistering of the lips, mouth, tongue, pharynx, and esophagus, the patient is in great distress and sits salivating excessively into a bowl. Dysphagia is obviously complete and within a day, unless treatment is instituted, the signs of acute dehydration appear.

*In the intermediate phase* with healing of the buccal and pharyngeal mucosa and separation of the esophageal slough, the patient temporarily can swallow again and has an unreal feeling that all will be well.

*In the final phase* as the developing stricture tightens, progressive dysphagia returns until, when, it is finally complete, everything swallowed is regurgitated.

**Less Severe Cases.** The dysphagia may be much less, the acute episode pass by be forgotten and remembered only when, years later, the slowly developing stricture is completely blocked by a bolus of poorly chewed food. This blockage from stricture may herald the appearance of a neoplasm and cannot lightly be ignored.

### MANAGEMENT

In this chapter detailed management will be confined to the acute and intermediate phases only.

**Acute Phase of Necrosis.** The five essentials of immediate management are to

- 1 Remove or neutralize the chemical agent, if possible
2. Relieve pain
- 3 Maintain nutrition
- 4 Counteract infection
- 5 Prevent stricture formation.

The following routine is advised for those present at the time of injury

- 1 *Encourage vomiting* by putting a finger or spoon in the throat. Also *identify and neutralize the chemical agent swallowed and administer the correct antidote*. To be of any value at all, as Krey has shown, administration of antidote must be immediate. Most cases are seen too late—possibly some hours after the injury.



- 2 *Pain* can be lessened by morphine or pethidine injections in doses graded according to age. Emollients such as olive oil are also soothing. The patient must be hospitalized.
- 3 *Nutrition* If he is unable to swallow any fluid, the patient is placed on intravenous therapy given at the rate of 3 liters per day. Additional fluid, equal in volume to that of the saliva lost per day, is also given. If the patient is still able to swallow, then such bland nutritious fluid as egg and milk should be given.
- 4 *Prevent Stricture Formation* Antibiotics—penicillin and streptomycin—are given to combat the inevitable secondary esophageal infection that follows the burn and intensifies the formation of granulation tissue.

In an effort to lessen stricture formation in a 17-month old boy with an acute lye burn, Weisskopf (4) used 25 mg of cortisone and 500 mg of terramycin 6-hourly for 3 days, followed by cortisone twice daily until the twelfth day.

**FURTHER TREATMENT DURING THE EARLY PHASE** Personal experience has shown that esophagoscopy during the first 10 days has little to offer. The esophagoscope passed beyond the cricopharyngeus muscle merely confirms the presence of white, edematous mucosa through which it is quite hazardous to attempt to proceed.

The patient should be continued on intravenous therapy, and if there is no return of esophageal patency in five to seven days, a Stamm gastrostomy should be performed.

At this phase of necrosis, treatment aims at resting the damaged esophagus and—until separation of the sloughing mucosa occurs—leaving a patent esophageal lumen surrounded by its cylindrical ulcerating wall.

*It is at this stage—the time when ability to swallow by mouth returns, some 10 days after injury—that much can be done to prevent the otherwise inevitable stricture.* So often, however, any return of swallowing is treated as being synonymous with the return of a normal esophagus that this chance of worthwhile help is lost, and the preliminaries of tight stricture formation are left to run their course.

**Intermediate Phase of Ulceration.** Whether the patient, by the foregoing treatment, has been successfully tided over into this phase or is seen for the first time in it, treatment is the same and aims at

- 1 Assessing the state of the esophagus,
- 2 Preventing any further stricture formation.

**Investigations. ESOPHAGRAMS** Watery radiopaque solutions serve to show the length and level of any esophageal constrictions.

**ESOPHAGOSCOPY** This is the most important of the investigations, to show the nature and extent of the esophageal granulations. General anesthesia is to be preferred, and, if a Ryle tube or silk thread has previously been swallowed, the esophagoscope is threaded over it and then guided down to the developing stricture, which is then gently dilated. A wide-bore stomach tube, e.g. Size 30 F for adults, is left in situ (*Vide infra*).

**Surgical Treatment of Ulceration. IN ADULTS** In the presence of obvious ulceration, the most satisfactory form of treatment, which at the same time dispenses with daily painful dilatations, is the method of internal esophageal splinting devised by Leegaard (5). The simple principle is that a wide-bore stomach tube, running the whole length of the esophagus, acts both as a food passage for tube feeding and as an



Fig. 1 Intraesophageal splint—wide-bore stomach tube introduced via cervical esophagostomy

intraesophageal splint around which the inflammatory reaction runs its course without forming a tight stricture. Intubation therefore lasts approximately two months. As the upper end of the tube is introduced via a cervical esophagostomy *below* the cricoid pharyngeal sphincter, the functions of the pharynx are unaffected (Fig. 1).

**Method.** The cervical esophagus is exposed through a two-inch incision along the anterior border of the left sternomastoid muscle, and the plane between the carotid sheath laterally and the larynx and the trachea medially is developed. The middle thyroid veins and inferior thyroid artery are divided, and the upper esophagus is isolated just beyond the level of the cricoid cartilage. It is lifted with Allis forceps and its lumen is opened. If the stomach tube has been left in situ after esophagoscopy, it is now caught up and its proximal end drawn through the wound. If not, then a well-lubricated tube is inserted through the esophageal wall and pushed down into the stomach.

When the tube cannot be made to enter the stomach from above, the writer (6) has performed a temporary gastrostomy, pushed a tube up the esophagus into the mouth, and thereafter attached a second tube to it from above and drawn this down into the stomach. The upper free end is then delivered through a left cervical esophagostomy as already mentioned.

The cervical wound is lightly packed with sterile paraffin gauze, and the tube is spigotted and strapped to the side of the neck. The great advantage of this method is that it achieves the aims of both gastrostomy and repeated bouginage by one maneuver.

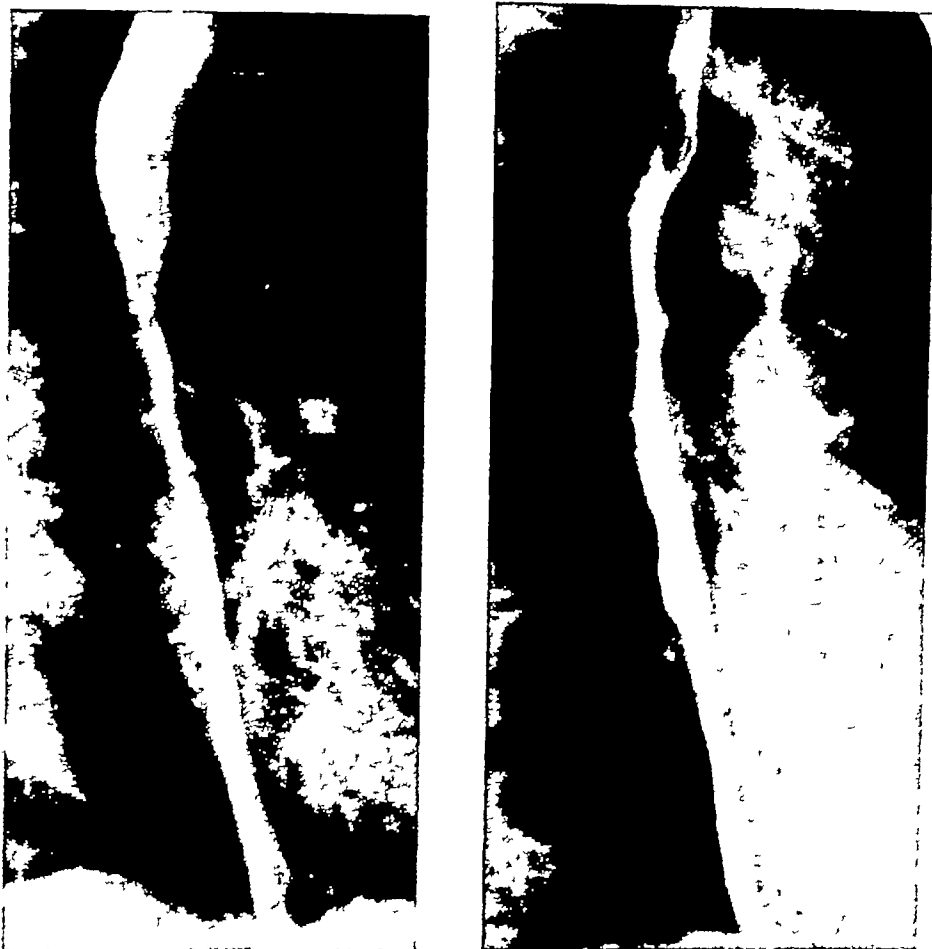


Fig 2 Left, Esophageal outline after removing esophageal splint Right, Esophageal outline 18 months after such treatment, showing adequate lumen

*Postoperative Management* The cervical wound is covered with absorbent dressings to mop up any leakage of saliva. Antibiotics—already commenced—are a further safeguard against cervical infection. High caloric fluid diet is given through the tube and a strict fluid balance and weight chart kept. After every feeding, water is run through and the tube carefully cleaned.

Changing of the tube for cleansing or because of blockage offers no hazard provided an identical one is prepared and properly lubricated ready to slip into the esophagus the moment the other is withdrawn. Initially, changing the tube may be necessary on two or three occasions at close intervals, but later it is required only weekly. If necessary, wider bore tubes can be introduced.

After two months with the tube temporarily removed, the esophagus is rechecked by an esophagram and esophagoscopy (Fig 2, left). If the inflammatory process has now subsided, the tube is discarded and the patient taught daily self-bouginage. If it is certain that the bougie passes into the stomach, the patient is discharged but should be seen first at weekly, and later at fortnightly, intervals for recheck. On each occasion, the patient passes the bougie in the presence of the surgeon. If there is a tightening of the stricture, redilatation is performed via esophagoscopy.

*Results.* Three adult cases, which the author treated by this means, are free from stricture and are swallowing normally from 2 to 10 years afterward.

**IN CHILDREN.** The foregoing regime has a great deal to recommend it, even with children, it gave excellent results in Leegaard's (5) hands. In milder cases, a re-

tamed catheter was introduced through the nose in severe cases external esophagostomy was used.

The alternative is either repeated dilatation from above—with or without anesthesia—or gastrostomy swallowing of a thread and daily dilatation with Tucker bougies. The ease of handling these patients by esophageal splinting makes this method preferable whenever possible.

**Final Phase of Stricture Formation.** The management of established strictures with the problems of repeated dilatation or esophageal replacement is beyond the scope of this chapter. The reader is referred to the author's article (7) for a summary of the principles involved or to the articles of Fatt (8) Marchand (9) and Carver and associates (1) for details of their large experience.

### CONCLUSIONS

Having treated many of these patients at varying stages in their illness, the author wholeheartedly supports Fatt and associates in their plea that "this is a disease which we should never have to treat, for it is the needless product of a society which continues to permit the free sale of caustic soda at a time when inexpensive and harmless detergents are readily available."

When the lesion is present, the sooner it is seen by one who understands the pathologic process involved, the better. If treated early by adequate internal esophageal splinting the hazards of late stricture formation are largely prevented and easier swallowing is assured.

### REFERENCES

1. Carver G. M., Sealy W. C. and Dillon, M. L. Management of alkali burns of the esophagus, *J.A.M.A.*, 160 1447 1956.
2. Krey H. On the treatment of corrosive lesions in the oesophagus. *Acta oto-laryng.*, Supp. 102 1-49 1952.
3. Boshier L. H., Burford, T. H. and Ackerman, L. The pathology of experimentally produced lye burns and strictures of the esophagus, *J. Thoracic Surg.*, 21 483 1951.
4. Weiskopf A. Effects of cortisone on experimental lye burn of the esophagus, *Ann. Otol., Rhin. & Laryng.*, 61 681 1952.
5. Leegaard, T. Corrosive injuries of the oesophagus with particular reference to the treatment of acute corrosive oesophagitis, *J. Laryng. & Otol.* 60 389 1945.
6. Borrie, J. Management of corrosive burns of the oesophagus, *Australian and New Zealand J. Surg.*, 25 62, 1955.
7. Borrie, J. In *Maingot's The Management of Abdominal Operations*, 2nd ed., London, H. K. Lewis & Co., Ltd. 1957 Vol. 1 p. 522.
8. Fatt, L., Marchand, P., and Cranshaw G. R. Treatment of caustic strictures of the esophagus, *Surg., Gynec. & Obst.*, 102 195 1956.
9. Marchand P. Caustic strictures of the esophagus, *Thorax*, 10 171 1955.

## SPONTANEOUS PERFORATION OF THE ESOPHAGUS

**Historical Note.** In 1724, the Dutch physician Boerhaave (1) first brilliantly recorded the onset, fatal course and autopsy findings of spontaneous perforation of the normal esophagus of the illustrious Baron de Wassenaer, Lord High Admiral of the Dutch Republic. Boerhaave's account is summarized by Barret (2). In 1877, the lesion was further described by Fitz (3) of Boston, and in 1914, it was comprehensively reviewed by Walker (4). In 1944, Collis (5) attempted suturing the rent, but his patient died from necrosis of the mediastinal tissue. In 1946, Barrett (6) first achieved successful repair.

### PATHOLOGY

Ninety per cent of the cases occur in men. Though Anderson (7,8) found the youngest reported was 2 years old and the oldest 75, in the 50 cases reviewed, most occurred in the fifth decade. The writer's oldest patient was aged 81 years.

Regarding the status of the esophagus prior to rupture, the mucosa and musculature were essentially normal, therefore, the patients who are seen fall into two groups:

- 1 *Rupture from Increased Intraesophageal Pressure.* Most commonly the lesion is mechanical in origin and due to a suddenly increased intraesophageal pressure that stretches and bursts the esophageal wall. Vomiting is the most common cause, especially the violent retching that follows excessive eating or drinking. Rupture also occurs from many other causes than vomiting, as from straining at defecation, or from the indirect trauma of abdominal or thoracic crush injuries. The writer (9) has also reported it from vomiting initiated by a duodenal ulcer, and has successfully treated it when initiated by hyperemesis gravidarum.
- 2 *Rupture with Apparently Normal Intraesophageal Pressure.* This occurs without vomiting or other similar cause. This is the true "spontaneous rupture" and can occur when the intraesophageal pressure is within normal physiologic limits. It is seen clinically in the weaker esophagus of the elderly. Anderson reported that 8 of 9 patients with this history were aged 53 years or more, a finding supporting Burt's (10) conclusions that an adult esophagus over 50 years of age will rupture with far less pressure than one from a child or young adult.

The site of the lesion is most commonly the relatively unsupported lower third of esophagus, the weakest esophageal segment (11). The left side is usually affected, accounting for 60 of 62 cases in Mackler's (12) series.

There is usually a single vertical rent varying from 2 to 8 cm long. Rarely transverse tears have been described (7) (1952) as well as complete disruption of the tube (1). Depending on the force with which the vomitus has been ejected through the rent, it may remain extrapleural and spread in the posterior mediastinum and extrapleural layers, causing mediastinal emphysema or it may rupture into one or other pleural cavity, causing hydropneumothorax, with or without tension effects. This rapidly progresses to pyopneumothorax.

In a series of five cases, the writer has had two that ruptured only into the posterior mediastinum, two into the left pleural cavity and one into the right pleural cavity.

Perforations of both the middle and upper thirds of the esophagus (13-14) have also been reported.

### CLINICAL FEATURES

**Symptoms.** There is usually a history of violent retching and vomiting especially after heavy eating or drinking or following other causes of vomiting already mentioned. The vomitus may be blood stained (9). The patient may, in fact, unwittingly precipitate his lesion by consciously trying to suppress his vomit. The lesion should also be remembered as a possibility when treating any severe abdominal or thoracic trauma (15).

Typically the patient is seized with an agonizing retrosternal pain which spreads over the affected hemithorax and radiates to the shoulder. It is aggravated by deep breathing and lessened by sitting up. The patient often describes a bursting sensation and is convinced he will die. Because of the hydropneumothorax and reduced pulmonary ventilation, there is severe dyspnea and cyanosis, both of which are aggravated by any increasing intrapleural pressure and mediastinal emphysema. There may also be voice changes. Severe thirst is common, and any drinking of water only increases the pain.

**Physical Signs.** Following intrapleural rupture, these patients are usually critically ill, often profoundly shocked, dyspneic, and cyanosed. There is usually epigastric guarding and tenderness. The chest signs are those of hydropneumothorax, with or without displacement of the trachea and apex beat. In at least half the cases there will be telltale subcutaneous emphysema appearing at the suprasternal notch and spreading over the neck, head, arms, and trunk.

When the pleura is intact, suprasternal cervical emphysema may be the only physical sign, while occasionally no physical signs at all are detected.

**Diagnosis.** This should be strongly suspected from the history and physical findings, especially by Mackler's triad (12) of vomiting, low thoracic pain, and cervical emphysema. It is confirmed by x-ray examination.

**ROENTGENOGRAMS.** Anteroposterior and lateral chest films are taken no matter how seriously ill the patient may appear, for today it is a fallacy to believe anyone is too ill for a portable x-ray film, especially when the lesion, if undiagnosed, is so rapidly fatal.

The roentgenograms confirm the presence of a hydropneumothorax and may show mediastinal emphysema outlining the aortic arch as a crescentic shadow (Figs 1 and 2).



Fig 1 Perforation into left pleural cavity Left hydro-pneumothorax and mediastinal emphysema following vomit after drinking bout Thoracotomy and suture-healing (From Borrie, J, in Maingot's Management of Abdominal Operations, 2nd Ed , 1957 Courtesy H K Lewis & Co Ltd ,London )

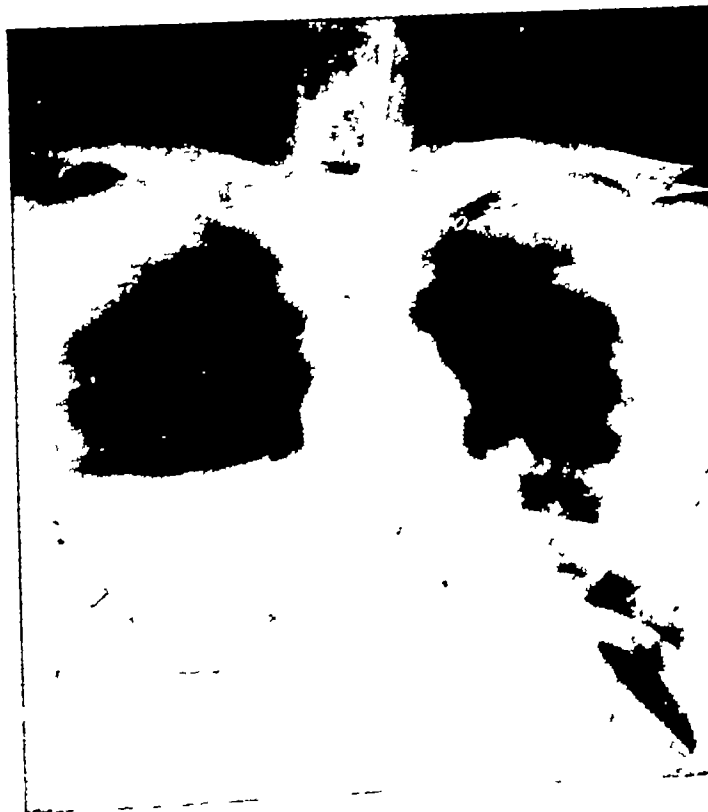


Fig 2 Perforation into right pleural cavity Hydro-pneumothorax in patient with history of duodenal ulcer, pain following vomiting, laparotomy proved fruitless Thoracotomy and suture were done, leakage developed, followed by empyema Drainage was instituted, healing followed (From Borrie, J , in Maingot's Management of Abdominal Operations, 2nd Ed , 1957 Courtesy H K. Lewis & Co Ltd , London )



Fig 3 Perforation into posterior mediastinum. Lipiodol swallow showed extraesophageal extravasation of radiopaque oil (arrow) Thoracotomy and suture were done healing followed. (From Borrie J in *Malingot's Management of Abdominal Operations*, 2nd Ed., 1957 Courtesy H K. Lewis & Co. Ltd., London.)

If doubt still exists the swallowing of radiopaque oil will show the perforation and will reveal dye outside the esophageal wall. This method is strongly recommended, especially when the mediastinal pleura is thought to be intact (Fig. 3)

Because these patients are often misdiagnosed as acute abdomen and subjected to needless laparotomy the author believes it is important that all x ray films of the "acute abdomen" be taken with the patient sitting up and positioned to include the lower half of the chest (9)

**DIAGNOSTIC CHEST ASPIRATION** As the fluid may be brownish or puslike in color its true significance can be underestimated The fluid is acid to litmus paper

**Differential Diagnosis.** Though the clinical and radiologic sequence of vomiting, pain, pleural fluid, and mediastinal emphysema is typical these cases are frequently misdiagnosed as coronary thrombosis rupture of a duodenal ulcer or gallbladder, acute pancreatitis, mesenteric thrombosis, or volvulus Failure to consider the possibility of a ruptured esophagus is the usual cause of these errors

**Prognosis.** Spontaneous perforation of the esophagus is a lethal disorder especially because of the shock, hemorrhage and tension pneumothorax that accompany it In 1948 Kinsella and associates (16) in a review of 53 cases, found that

13 died in less than 12 hours

24 died in less than 24 hours

8 died in less than 48 hours and

only 8 survived beyond 48 hours.



## TREATMENT

Admittedly, Graham (17), Anderson (7) and Susman (18) have shown that it is possible to save some patients by pleural or mediastinal drainage without esophageal suture, and that that is all one can offer those fortunate enough to survive for 48 hours untreated. But having repeatedly seen the quick return to full activity following emergency thoracotomy and suture of the perforation, the writer is convinced such is the right and proper course to take.

**Preoperative Treatment.** This follows the general lines stated in Chapter 3

- 1 The first and most important first-aid step is to relieve any tension pneumothorax by water-seal drainage. After this initial treatment, one of the writer's patients was able to be transported 70 miles for further operation.
- 2 All fluid by mouth is stopped, including salivation, by giving atropine 0.6 mg and morphine 15 mg.
- 3 Intravenous therapy, including blood transfusion, is commenced.
4. A Levin tube is passed into the stomach and constant low pressure suction applied.
5. Antibiotic therapy is commenced.

With this regime the patient should improve rapidly.

**Operation.** Preliminary esophagoscopy can be done but is not essential, especially if a water-seal drain has already been inserted.

Thoracotomy is performed through the eighth rib bed. A rib spreader is inserted, fluid and fibrin clot removed, and the pulmonary ligament divided. The mediastinal pleura usually has a shaggy rent which is enlarged. If, however, the mediastinal pleura is found to be intact, it is incised vertically. The esophagus is encircled with a finger and held retracted with a catheter sling. The edges of the rent are carefully trimmed with scissors, stay sutures inserted as required, and the rent closed with interrupted 4-0 silk sutures (Figs 4 and 5). If inflammation is already present, stainless steel sutures are desirable. The pleural cavity is thoroughly cleansed and irrigated with saline. The lung is reinflated, and the chest wall is closed over water-seal drainage with interrupted catgut sutures for the deep layers and nylon sutures for the skin.

**Postoperative Care.** In general, postoperative care follows the lines discussed in Chapter 3.

**FEEDING.** *When the rent is small—3 cm or less—*intravenous fluid is maintained for four days, after which time the patient may take milk, glucose, and water by mouth (see Acquired Esophageal Perforation, Chapter 19). All solid food is withheld for two weeks. Intercostal drainage is maintained until the tube becomes blocked—usually in two or three days.

*When the rent is larger and the degree of pleural contamination greater,* then all oral feeding is stopped, and a jejunostomy is performed and maintained until healing is complete, as confirmed by esophagrams.

**Complications.** *Leaking Suture Line.* This may develop between the seventh and tenth days, especially if oral feeding has been hastened. This emergency is treated by intercostal drainage with continuous suction and jejunostomy. The intercostal drainage must be maintained until the fistula closes, as judged by esophagrams with fluid media.

*Empyema* may also develop without leaking of the suture line. It should be treated as outlined in Chapter 9.

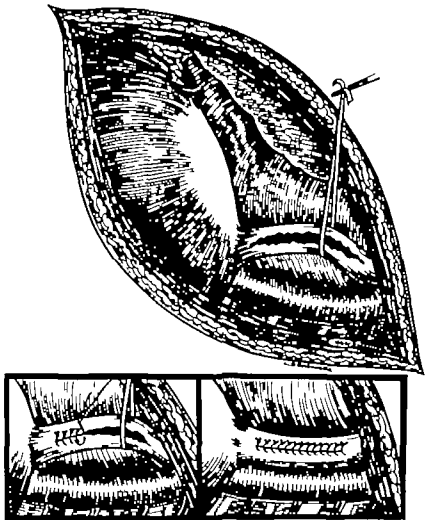


Fig. 4   Technic for repairing spontaneous perforation of esophagus into left pleural cavity

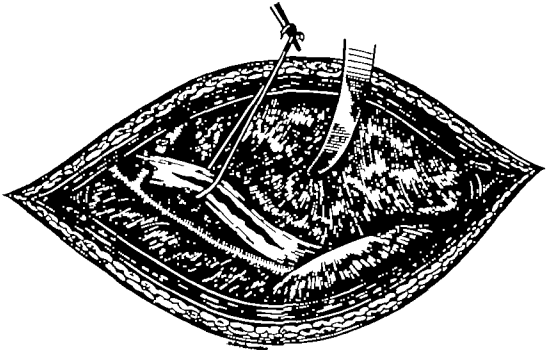


Fig. 5   Technic for repairing spontaneous perforation of esophagus into right pleural cavity

**Results.** Derbes and Mitchell (19) in 1956 found that, if surgery is not performed, only 25 of 71 patients survived for 24 hours; 8 lived to the end of the second day, and none lived longer than one week

Of a series of 55 patients who have been operated on to date, however, 35 have survived. It is therefore obvious that greater awareness and more astute diagnosis are required if these results are to be improved.

### CONCLUSIONS

This acute surgical emergency should always be kept in mind, especially when there is a history of vomiting followed by severe chest pain. Chest roentgenograms or esophagrams confirm the diagnosis. Treatment is by emergency intercostal water-seal drainage to reduce intrapleural tension, followed by emergency thoracotomy and suture.

### REFERENCES

- 1 Boerhaave, Hermann *Atrocis, nec descripti prius morbi historia. Secundum medicae artis leges conscripta*, Lugd. Bat. Boutesteniana, 1724. English translation: Bull. M. Library A., 43:217, 1955.
- 2 Barrett, N. R. Spontaneous perforation of the oesophagus. Review of the literature and report of three new cases, *Thorax* 1:48, 1946.
- 3 Fitz, R. H. Rupture of the healthy oesophagus, *Am. J. Med. Sci.*, 73:17, 1877.
- 4 Walker, I. J. Spontaneous rupture of the healthy esophagus, *J. A. M. A.* 62:1952, 1914.
- 5 Collis, J. L., Humphreys, D. R., and Bond, W. H. Spontaneous rupture of the oesophagus, *Lancet*, 2:179, 1944.
- 6 Barrett, N. R. Report of a case of spontaneous perforation of the oesophagus successfully treated by operation, *Brit. J. Surg.*, 35:216, 1947.
- 7 Anderson, R. L. Rupture of the esophagus, *J. Thoracic Surg.*, 24:369, 1952.
- 8 ——— Spontaneous rupture of the esophagus, *Am. J. Surg.*, 93:282, 1957.
- 9 Borrie, J. Spontaneous rupture of the oesophagus. Report of three successful cases treated surgically, *Brit. M. J.*, 1:23, 1955.
- 10 Burt, C. A. V. Pneumatic rupture of intestinal canal with experimental data showing the mechanism of perforation and the pressure required, *Arch. Surg.*, 22:875, 1931.
- 11 Mallory, G. K., and Weiss, S. Hemorrhage from laceration of the cardiac orifice of the stomach due to vomiting, *Am. J. Med. Sci.*, 178:506, 1929.
- 12 Mackler, S. A. Spontaneous rupture of the esophagus: an experimental and clinical study, *Surg., Gynec. & Obst.*, 95:345, 1952.
- 13 Nanson, E. M., and Walker, R. M. Partial spontaneous rupture of the oesophagus, *Brit. J. Surg.*, 40:574, 1953.
- 14 Russell, J. Y. W. Spontaneous perforation of the oesophagus, *Brit. J. Surg.*, 40:312, 1953.
- 15 Borrie, J. The management of major thoracic trauma, *Australian and New Zealand J. Surg.*, 26:229, 1957.
- 16 Kinsella, T. J., Morse, R. W., and Hertzog, A. J. Spontaneous rupture of the esophagus, *J. Thoracic Surg.*, 17:613, 1948.
- 17 Graham, E. A. Editorial comment, *Yearbook of General Surgery*, 1944, 382.
- 18 Susman, M. P. Rupture of the oesophagus, *Australian and New Zealand J. Surg.*, 22:273, 1953.
- 19 Derbes, V. J., and Mitchell, R. E. Rupture of the oesophagus, *Surgery*, 39:865, 1956.

## ACQUIRED ESOPHAGEAL PERFORATION

As untreated esophageal perforations inevitably lead to acute suppurative mediastinitis, these two lesions are logically considered in relation to each other

## PATHOLOGY

The most common cause of acquired esophageal perforation is faulty passing of an esophagoscope or of dilating bougies. Less common causes are penetrating injuries of the neck, foreign bodies during ingestion or extraction, or perforation of a carcinoma, diverticulum, or "peptic" esophageal ulcer. Bell (1) in 1956 reported an incidence of 0.71 per cent in 35,000 gastroscopies. Because of its anatomy the esophagus is especially vulnerable both at its origin and lower end, and, with the increasing use of diagnostic endoscopy, there is ample evidence of a rising incidence of such injuries.

**In the Neck.** Of acquired esophageal perforations, 90 per cent occur in the cervical esophagus usually through one of the pyriform fossae. This is due to failure of the operator to appreciate that the cricopharyngeal sphincter is not normally relaxed, even by moderate anesthesia, that esophagoscopy requires utmost gentleness and that a bougie passed as an obturator through the sphincter is a very useful guide to the lumen. If the lesion is not recognized and the esophagoscope further advanced, it can readily pass on through the mediastinal pleura into either pleural cavity to cause a pneumothorax with or without tension effects. This vallecular perforation is usually an irregular transverse mucosal tear.

Alternatively, when the neck is extended and the lower cervical vertebrae bowed forward during the passage of the esophagoscope, the esophageal wall may split vertically just below the cricopharyngeal sphincter without the operator realizing it. Overstreet (6) reported five such cases in eight endoscopic perforations. Surgical emphysema is usually obvious within one or two hours and cervical induration within a day. This can become localized to a cervical abscess or more usually spread into suppurative mediastinitis within four to five days. If either the mediastinal pleura or pericardium is also perforated, a pyopneumothorax or suppurative pericarditis will form rapidly.

**At the Cardia.** The remaining 10 per cent of perforations occur mostly at the cardia as it passes forward and to the left into the stomach. The perforating bougie usually passes straight down either into the lesser sac or the bare area of stomach where local cellulitis rapidly advances to subphrenic abscess formation. Midesophageal perforations are usually due to foreign bodies.

## CLINICAL FEATURES

**In the Neck.** During esophagoscopy perforation may be recognized by the operator noting blood and torn mucosa as the esophagoscope is withdrawn. Where it has entered the pleural cavity, every respiration produces unwelcome gushes of air through its lumen. In one case on which the author called to aid the patient,



Fig 1 Cervical emphysema following instrumental perforation of esophagus



Fig 2 Lateral film of neck showing forward displacement of larynx and pharynx 48 hours after esophagoscopy, 0.5 cm vertical perforation through cricopharyngeal sphincter was drained, and healed in one week

and left lung were clearly visible through the esophagoscope. Alternatively, immediately after swallowing a sharp fish or meat bone, or on recovery from anesthesia for esophagoscopy and attempting to drink, the patient complains of an ominous retrosternal pain aggravated by every swallow.

No matter what the cause, the subsequent events are the same. Cervical emphysema, which may be minimal or massive, develops rapidly. Within a day, the neck is indurated and brawny from cellulitis of the fascial planes. There is a concurrent fever, tachycardia, and leukocytosis. Untreated, the infection advances. It may localize as a cervical abscess or spread to cause mediastinitis which is usually fatal within a week.

**At the Cardia.** A typical history is one of difficulty in dilating a benign or neoplastic stricture during esophagoscopy, followed by sudden, surprising ease and telltale blood staining. On recovery from anesthesia, the patient complains of severe persistent epigastric pain, from which he can get no relief and which is also aggravated by swallowing even of saliva. There is epigastric tenderness and rigidity, but, as the perforation does not open into the greater sac, no rectal tenderness. Within a few hours, the temperature and pulse rate are moderately raised.

No matter how slight, these symptoms are significant and must not be ignored. As it is known that even in the best clinics esophageal perforation after esophagoscopy does rarely occur, all patients so investigated should remain in hospital for a short period of postoperative observation following this procedure.



**Mortality.** Perforation of the esophagus is a serious emergency. In his series of 69 patients treated between 1925 and 1947 Jemerin (2) reported that 44 recovered and 25 died—a gross mortality of 36.2 per cent. Prior to 1936, the mortality was 77.3 per cent and after 1936, 17 per cent. He noted that before 1936, operation was performed less than 1 week after perforation in only 3 of the 11 patients treated surgically, whereas after 1936, 35 of the 42 operations were performed less than 1 week after perforation, and 20 of these within 2 days.

As with all other perforations of the gut, *time matters*. Once the diagnosis has been made, only one course remains. As it is not the perforation itself that kills but rather its effects—namely, cervicomediatinitis, pericarditis, or tension pneumothorax—*prompt, adequate surgery offers the only chance of success*.

### MANAGEMENT

**Early Cervical Perforation.** **PREOPERATIVE MEASURES.** An intravenous infusion is commenced, general anesthesia induced, and the patient's trachea intubated. If the roentgenograms have not revealed the site of perforation, a further careful esophagoscopy may be carried out.

**PRINCIPLES OF OPERATION** Depending on the site of the perforation, an oblique skin incision is made along the anterior border of the sternomastoid muscle from the upper border of the thyroid cartilage to just above the clavicle. This is deepened so that the sternomastoid muscle and carotid sheath are retracted laterally, and the viscera—larynx and trachea, pharynx and esophagus—are retracted medially. The middle thyroid veins and inferior thyroid artery are divided. The esophagus is gently lifted forward and the perforation visualized (Fig. 5).

If the perforation is large, this is closed by a series of interrupted chromic 1-0 catgut sutures. If the perforation is small or in an infected area, it can be left alone. The important step, however, is to drain with a Penrose rubber tube, pack the wound lightly with petrolatum gauze, and loosely approximate the skin.

**POSTOPERATIVE CARE** As the wound is potentially infected, prophylactic injections of 500,000 units of penicillin and 0.5 gm. of streptomycin twice daily are given for the first week. The drain is removed on the third day, and the packing and skin sutures are removed two or three days later.

**Feeding** presents a variety of problems. Come what may, the patient continues to swallow his saliva. Intravenous therapy of isotonic glucose solution is continued at the rate of 3 liters a day for three days.

When the perforation has been sutured promptly and well, the patient may sip sterile water after 24 hours and take up to 60 ml. an hour for the next two days. This is supplemented by milk from the fourth day onward; thereafter, a soft gastric ulcer type of diet may be given. As suture lines have been known to break down, solid foods are not advised until after 14 days.

When, however, it is felt the suture line should be rested further and nothing be given by mouth, then either

1. The intravenous fluid is continued at the rate of 3 liters per day for three more days, or
2. A Levin tube is passed via the nose and swallowed, and the patient is fed a high caloric fluid diet through it.
3. Rarely, *gastrostomy* is indicated, especially if the causative lesion is an inoperable esophageal carcinoma or a benign esophageal stricture that may later require retrograde dilatation.

**Treatment for Extramucosal Intramural Perforations.** The treatment is shown in the following case

**CASE REPORT** The author accidentally made this lesion with a size 12 French bougie while attempting for the third time to dilate a tight esophageal web. Esophagram findings are seen in Figure 4

**Management** On May 14 1957 right thoracotomy was performed. There was a free pleural cavity. The azygos arch was divided and the esophagus above it isolated. There was no surrounding induration or cellulitis. It was therefore decided to continue with retrograde dilatation of the stricture. When however the esophagus

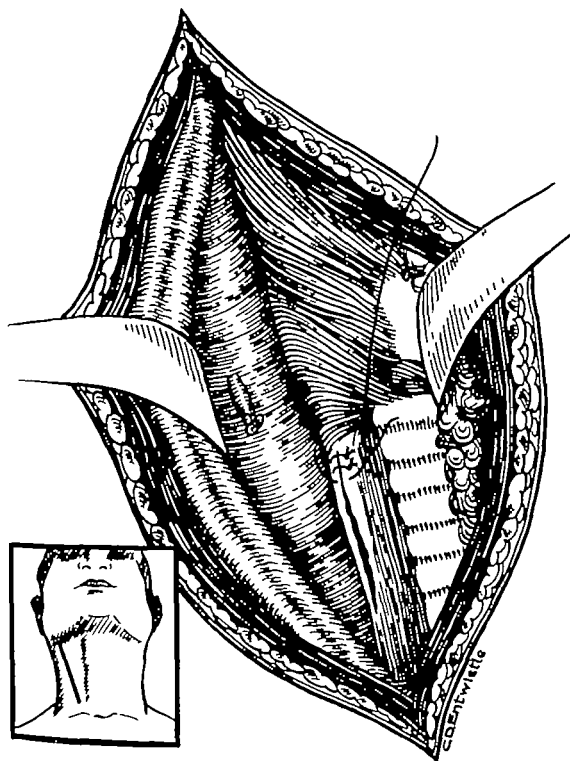


Fig. 5 Principles of operative repair of instrumental perforation of cervical esophagus.



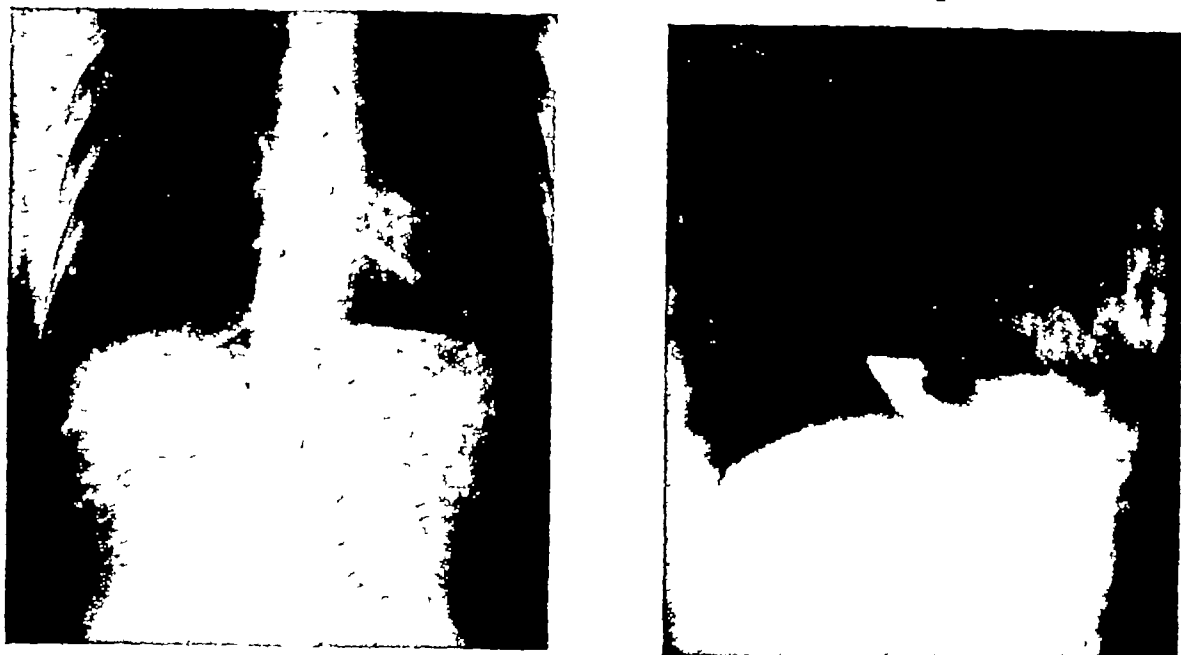


Fig 6 Perforation of cardiospasm at cardia from too-vigorous dilatation with Negus hydrostatic dilator, epigastric pain. Esophagrams 24 hours after dilatation left, opaque medium lying in bare area of stomach, right, lateral film showing no change one hour later Treated by thoracotomy and suture (Heller operation) with gastrostomy, recovery (From Borrie, J, Maingot, R, Management of Abdominal Operations, 2nd Ed, 1957, Vol 1, p. 500 Courtesy H K. Lewis & Co Ltd, London)

geal musculature was vertically incised, whitish radiopaque dye oozed forth and the true nature of the perforation was recognized. The underlying thoracic esophageal mucosa was next opened and the stricture dilated by passing a series of Timon catheters in retrograde fashion. It was not possible to suture the mucosal perforation above. The thoracic esophageal opening was next resutured in two layers with chromic catgut sutures, and the chest wall closed over water-seal drainage. Because of the stricture and possible need for further retrograde dilatation, a Stamm gastrostomy was performed.

The patient made an uneventful recovery and responded to subsequent peroral dilatation with an esophagoscope. The gastrostomy tube was removed three weeks after operation.

**Early Thoracic Perforations. OPERATIVE MEASURES** After definition of the lesion by esophagrams, thoracotomy is performed on the appropriate side and the esophagus isolated. Further procedures depends on the findings.

If the perforation is *small* and caused by the sharp edge of a foreign body, the lumen is incised vertically, the foreign body removed, and the wall resutured in two layers.

If the esophageal wall has been *lacerated* and in part divided, the raw edges are trimmed back and end to end suture carried out.

If there is a specific endoesophageal lesion, it is treated according to its nature, e.g.

1. A cardiospasm ruptured by a hydrostatic dilator requires suture and performance of a Heller operation (3), as previously described by the author (Fig 6).
2. An operable carcinoma requires resection and esophagogastrostomy. McBurney and associates (4) in 1953 reported five cases successfully resected.

If, however, the injury is too extensive or the patient is too debilitated to withstand a major operation, the aim is to save life by

- 1 Mobilizing the cervical esophagus to the surface as a cervical esophagotomy,
  - 2 Closing the lower esophageal stump and performing a Stamm gastrostomy
- At a later date planned esophagogastrostomy can be performed by taking the stomach to the neck through the left pleural cavity and resecting the anterior end of the left first rib and a corresponding two inches of the left clavicle (5-6)

**RESULTS.** In three instrumental perforations referred to the author within three hours after perforation immediate operation permitted satisfactory suture in a clean field. In a fourth, recognized 48 hours afterward, cervical drainage alone was done. All these patients recovered.

### CONCLUSIONS

The possibility of perforation occurring even in experienced hands during esophageal instrumentation must always be kept in mind. When the condition is recognized there is only one satisfactory treatment—immediate suture. Recovery is then the rule. When the condition is recognized late, drainage of the cervical or subphrenic abscess offers the only chance of survival.

### REFERENCES

- 1 Bell, J. W., Beakin, C. A., Starkey, G. W. B. Mediastinal abscess following instrumental perforation of the esophagus, *Am. J. Surg.*, 91:999, 1956.
- 2 Jemerin, E. E. Results of treatment of perforation of the esophagus, *Ann. Surg.*, 128:971, 1948.
- 3 Borrie, J. In Maingot, R. H. *Management of Abdominal Operations*, 2nd ed., London, H. K. Lewis & Co., Ltd., 1957. Vol. 1, p. 500.
- 4 McBurney, R. P., Kirklin, J. W., Hood, R. T., and Andersen, H. A. One-stage esophagogastratomy for perforated carcinoma in the presence of mediastinitis, *Proc. Staff Meet. Mayo Clin.*, 28:281, 1953.
- 5 Rapant, V., and Hromada, J. Surgical treatment of corrosive stenosis of the thoracic part of the esophagus by a single stage palliative operation, *J. Thoracic Surg.*, 20:454, 1950.
- 6 Overstreet, J. W., and Ochsmar, A. Traumatic rupture of the esophagus, with a report of 13 cases, *J. Thoracic Surg.*, 30:164, 1955.

# Mediastinal Emergencies

## 20

### SUPPURATIVE MEDIASTINITIS AND MEDIASTINAL EMPHYSEMA

#### SUPPURATIVE MEDIASTINITIS

**Introduction.** This serious lesion carries a high mortality and requires emergency drainage if life is to be preserved. The early literature was reviewed by Gaudiani (1) in 1916. The *indirect* or cervical approach was first used by Lurman (2) in 1876, when he successfully drained a large posterior mediastinal abscess containing 2.5 liters of pus. His method still has a place for cervical and superior mediastinal abscess. The *direct* or thoracic approach was described by Nassilow (3) in 1888, and, as it gives dependent drainage, is to be preferred, especially for lesions in the posterior mediastinum. When the lesion lies in the anterior mediastinum, anterior mediastinotomy is required.

#### PATHOLOGY

Mediastinitis is most commonly an infection of the posterior mediastinum and is always secondary to some other condition. It may arise by one of four possible routes:

1. Direct contamination from esophageal perforations (see Chapter 19),
2. Fascial plane extension,
3. Direct spread from lungs, pleura, or pericardium,
4. Spread by the blood or lymphatic streams from a distant abscess.

**Direct Contamination.** This is by far the most common cause for, as Neuhoﬀ (4) observed, over 50 per cent of cases of suppurative mediastinitis result from spontaneous or instrumental perforation of the esophagus, especially by an esophagoscope or bougies. This occurs most commonly in the cricopharyngeal region (Fig. 1). Less commonly it may arise from

1. Foreign bodies,
2. Perforation of a neoplastic or peptic esophageal ulcer,
3. Traumatic perforation from a stab or gunshot wound,
4. Spontaneous perforation;
5. Following breakdown of an anastomosis after esophageal surgery.

When the pleura or pericardium has also been perforated, empyema or suppurative pericarditis results.

**Fascial Plane Extension.** This comes almost invariably from cervical infection. As mentioned in Chapter 19, retropharyngeal cellulitis will often spread down into the posterior mediastinum and further infiltrate around the aorta and lung roots (Fig. 2).



Fig. 1 Lateral film of cervical abscess secondary to perforation of cervical esophagus. (From Borrie, J. in Maingot, R., *Management of Abdominal Operations*, 2nd Ed., 1957. Courtesy H. K. Lewis & Co., London)



Fig. 2. Admission posteroanterior and lateral chest films 10 days after esophageal perforation at esophagoscopy: mediastinal abscess, with hilar fluid level and right basal pneumonia. (From Borrie, J., in Maingot, R., *Management of Abdominal Operations*, 2nd Ed., 1957. Courtesy H. K. Lewis & Co. Ltd., London)



Fig 3 Anterior mediastinal abscess secondary to multiple staphylococcal abscesses including a right subphrenic abscess (Anterior and lateral views)

**Direct Spread from Lungs, Pleura, or Pericardium.** Rarely, this may occur when a paramediastinal lung abscess or empyema penetrates into the mediastinum

**Lymph Spread.** Rarely, too, infection of those areas with normal lymph drainage through the posterior mediastinum (e g the subphrenic or perirenal spaces, the pericardium, lungs, and esophagus) may cause suppurative mediastinitis, usually as a terminal event. It may also arise, even in the anterior mediastinum, from a septicemia based on multiple staphylococcal abscesses (Fig 3).

**Course of Infection.** Almost without exception, the infection is at first confined to either the superior or posterior mediastinum, as the process develops, it tends to form an abscess which may spread along either hilum into the lungs. In some cases, it will erode directly into the adjacent adherent lung to perforate into a bronchus (4)

As pus increases in quantity, however, it extends from the posterior into the middle and anterior mediastinum. With phlegmonous mediastinitis, pus may even extend downward to involve the retroperitoneal structures and peritoneum. As already mentioned, both pleural and pericardial cavities are vulnerable to infection either via a perforation or by direct spread.

### CLINICAL FEATURES

**Signs and Symptoms.** The signs and symptoms are essentially those of an advancing infection that follows an untreated esophageal perforation. Following the episode of perforation, however, there may be a transient but treacherous improvement.

The patient complains of dysphagia and pain, especially pain in the neck or

chest on swallowing, pain in the epigastrium or shoulder from diaphragmatic involvement, and pleuritic pain on inspiration. There are the general signs of toxic infection. In the neck, there may be cervical induration, possibly surgical emphysema, and suprasternal tenderness. The trachea is usually pushed well forward by the mediastinal infection and is occasionally deviated to one side. In the chest, unless the pleural or pericardial cavities have already been entered, there is a paucity of physical signs.

If a bronchomediastinal fistula has occurred, there may be a history of transitory remission after expectorating foul sputum. In the rarer abscess of the anterior mediastinum, Neuhof reports associated inferior vena caval obstruction. He points out that the diagnostic complexities of a prevertebral abscess perforating into the pleura or of a paravertebral lung abscess or empyema invading the mediastinum are usually only recognized at operation.

**Röntgenography.** Lateral cervical roentgenograms are invaluable in outlining an abscess and must be taken and interpreted promptly (Fig. 1). Posteroanterior and lateral chest films clearly show the effects on the mediastinum, lung, pleural, or pericardial cavities (Fig. 2). In the presence of air, a fluid level is obvious. Anterior displacement of the trachea is significant. Esophagrams will outline an esophageal perforation and the associated abscess cavity.

## TREATMENT

### *Prompt dependent surgical drainage is imperative*

**Preoperative Management.** Chemotherapy by injection is started at once, the patient receiving penicillin 2 mega and streptomycin 1 gm. a day. As these unfortunate patients lose weight rapidly, become profoundly debilitated, and develop a severe iron-deficiency anemia, full blood examination (including hemoglobin estimation, packed cell volume, white cell count, and blood grouping) is required. Blood transfusion is commenced. As sedatives may precipitate a terminal bronchopneumonia, atropine alone is given.

**Operative Procedures.** General anesthesia with tracheal intubation is advised.

**CERVICAL AND SUPERIOR MEDIASTINAL INFECTION.** When the infection is localized at the root of the neck, an oblique incision is made along the anterior border of the left sternomastoid muscle; the middle thyroid veins are divided; the visceral compartment of larynx and trachea, pharynx and esophagus retracted medially and the carotid sheath retracted laterally. The abscess, which usually extends behind the esophagus, is incised, the pus sucked away, and a Penrose tube drain and gauze pack left in situ. Thereafter a right posterior mediastinotomy through the bed of the third or fourth rib, as described by Adams (5), establishes counter drainage and avoids the aortic arch, its left subclavian branch, and the thoracic duct.

**POSTERIOR MEDIASTINAL INFECTION.** If local anesthesia is used, the patient sits on the side of the operating table with his arms supported on a Mayo table, as described under treatment of acute empyema (Chapter 9). If general anesthesia is used, however, he lies on one side with his head and trunk inclined upward at 30 degrees.

The level of the mediastinal abscess is carefully localized by checking rib levels on the chest films, and a left or right approach is made depending on which side shows the greater mediastinal widening and pleural reaction. For high abscesses the third or fourth ribs are chosen; for paravertebral ones the fifth or sixth; and for low mediastinal infection, the eighth rib usually suffices. The operative technic is the same.

described under rib resection for the treatment of acute empyema, except that usually only the posterior two inches of rib require resecting

After rib section, the mediastinum, which is hard and indurated, is carefully needled for pus, and sinus forceps are inserted into the abscess cavity. A soft rubber drain is inserted into the abscess and attached to a water seal. Because of adhesions, it is unlikely that the pleural cavity will be entered, but if so, then a water-seal drain is inserted into the base of the pleural cavity. A specimen of pus is cultured and antibiotic sensitivities determined.

**SUBPHRENIC INFECTION** If the esophagus has been perforated at the cardia and has been neglected, the resulting subphrenic abscess within the bare area of stomach is localized by needling and drained through the left eleventh rib bed.

**PLEURAL AND PERICARDIAL INFECTIONS** Their management is described in Chapters 9 and 22, respectively.

**Postoperative Management.** **GENERAL STATE.** Blood transfusion is continued on a slow drip until the hemoglobin level is restored to normal, and thereafter continued for 48 hours with glucose-saline infusion (4 per cent glucose and 0.2 per cent normal saline).

Broad spectrum antibiotics are given, using penicillin 1 mega and streptomycin 0.5 gm twice daily, and the schedule of antibiotics is adjusted when the specific sensitivity tests are completed. With effective drainage, antibiotics seldom need be continued beyond one week. Routine physiotherapy is required to improve breathing, coughing, arm movements, and ambulation.

**THE QUESTION OF GASTROSTOMY** Once the abscess has been drained and the danger point passed, the question of gastrostomy is raised. Almost without exception, this procedure is advisable as being the only sure way of resting the esophagus and encouraging healing of any esophageal perforation. Stamm gastrostomy through a left upper rectus-splitting incision is performed under local anesthesia. If the patient's condition will allow it, the gastrostomy can be established *immediately prior* to draining the abscess.

**DIET.** After a gastrostomy, a fluid diet can be given immediately, but is usually withheld for 24 hours, when water is given 1 oz per hour for 12 hours followed by a high caloric fluid diet. Strict fluid balance and weight charts are kept. The gastrostomy sutures are removed after one week.

**THE WOUND** Discharge of pus is at first copious and requires at least thrice daily dressings. Within a week, it lessens and the wound heals around the tube. This continues to drain until the esophageal fistula ultimately closes by granulation in four to six weeks. Closure is confirmed either by having the patient swallow methylene blue solution or by checking with esophagrams, using watery opaque media. Thereafter, the drainage tube can be gradually shortened and the sinus encouraged to heal from within outward by keeping the tube 2 cm shorter than the total sinus length. Finally, when the sinus is healed, the patient has further check esophagrams.

**THE ESOPHAGEAL LESION.** When the mediastinitis arises on a perforation during esophagoscopy, inspection of a removable malignant or benign lesion, once the infection has subsided, planned resection can be considered and performed. When, however, the lesion is a benign stricture from caustics or an esophageal web and subsequent dilatation from above proves difficult, the patient can swallow a strong linen thread. This is brought out of the gastrostomy wound with the aid of an esophagoscope inserted through the gastrostomy opening. Thereafter, the thread is tied to a set of Tucker bougies, and retrograde dilatation of the stricture is performed. This

is later changed to peroral dilatation, the gastrostomy tube is withdrawn, and the opening is allowed to heal.

**Results.** In 1941 Burnett (6) found a mortality rate in cases of undrained abscesses ranging between 60 and 80 per cent and for the group in which adequate drainage was established between 33½ and 40 per cent. In 1946 Adams reported successful recovery in seven patients all of whom had mediastinal drainage established within three hours of consultation and diagnosis. More than one drainage may be required. Bell and associates (7) in 1956 reported perforation of the thoracic esophagus by a flexible gastroscope. The patient recovered following drainage of the pericardium and of a huge mediastinal abscess by combined anterior and bilateral posterior mediastinotomies.

## MEDIASTINAL EMPHYSEMA

Although minor degrees of mediastinal emphysema may occasion no alarm, there are times when it is so massive and under such tension that it becomes a real emergency.

### PATHOLOGY

Air may enter the mediastinum by any of four routes

- 1 From the interstitial tissue of the lung
- 2 From the mediastinal viscera—e.g. a perforated trachea, bronchus or esophagus
- 3 From the neck
- 4 From pleural or retroperitoneal spaces

Most cases seen follow pulmonary alveolar rupture which causes interstitial emphysema of the lung. Macklin and Macklin (8) have shown experimentally that interstitial air bubbles migrate toward the hilum of the lung, and that once started this process becomes aggravated, each breath driving more air into the mediastinum. If the condition is unrelieved, increasing compression of the pulmonary alveoli and vessels may lead to death from asphyxia.

**Causes.** Interstitial pulmonary emphysema may arise in the following ways:

- 1 *Spontaneous Rupture of Alveoli* This is not uncommon. It was reported in almost half of Fagin and Schwab's (9) 39 cases of mediastinal emphysema.
- 2 *Increase of Intrapulmonary Pressure* This may arise from excessive straining with the glottis closed, from positive pressure anesthesia, from tracheal or bronchial occlusion, or from lung disorders such as asthma (10), bronchitis or pneumonia (11, 12). It may also arise during bronchoscopy even in children (13). In all of these instances it can be severe enough to require relief by cervical mediastinotomy.
- 3 *Chest Trauma* As mentioned in Chapter 5, major thoracic trauma may lead not only to severe mediastinal emphysema from rupture of pulmonary alveoli but also to an associated bronchial rupture or lacerated lung that will require thoracotomy. Scannell's case (14) required suturing of a torn right main bronchus and resection of a torn upper-lobe segment.
- 4 *Perforation of the Trachea, Main Bronchus or Esophagus* In addition to the foregoing causes, mediastinal emphysema may result from perforation of the trachea, main bronchus or esophagus.



5. *Operations on Neck or Dorsal Abdominal Wall* Rarely, emphysema may complicate cervical operations such as thyroidectomy (15) or tracheotomy (16), or follow the diagnostic introduction of air into the retroperitoneal region (17) and require surgical relief.

From the mediastinum, air may spread subcutaneously over the entire body, pass into one or both pleural cavities to cause pneumothorax, or pass through the diaphragm into the retroperitoneal planes.

### CLINICAL FEATURES

**Signs and Symptoms.** These may be summarized as (18):

- 1 Retrosternal pain radiating over the chest from distension of mediastinal tissue;
- 2 Subcutaneous and retroperitoneal emphysema;
- 3 Obliteration of cardiac dullness,
- 4 Peculiar sounds over the heart, especially loud, coarse, bubbling or crunching sounds with each contraction of the heart—"Hamman's Sign;"
5. Signs of increased mediastinal pressure, dyspnea, cyanosis, engorged veins leading to right-sided heart failure,
6. Pneumothorax.

In addition, anteroposterior roentgenograms may show the characteristic outlining of the aortic arch and pericardium, while lateral ones are of value in revealing air lying between the sternum and heart

### TREATMENT

Provided there is no serious underlying cause such as esophageal perforation, small amounts of air in the mediastinum require only observation, especially when the patient's pulse and respiration rate are not deteriorating nor the emphysema extending. Any pneumothorax requires relief with water-seal drainage. When, however, there is any respiratory embarrassment, the trapped mediastinal air must be released surgically.

**Technic of Operation.** Under local anesthesia, a transverse incision is made above the suprasternal notch, the pretracheal muscles are separated in the midline, and the trachea exposed. The trachea is digitally followed down into the mediastinum as far as the aortic arch. Air freely bubbles up. A Penrose drain is placed deeply into the wound and the skin loosely approximated. The skin sutures need not be tied until later. In Wurstmann's case, relief was obtained only by division of the sternum.

**After-Care.** If the operation relieves the cyanosis and dyspnea and there is no more serious underlying lesion, patients who have had cervical mediastinotomy require no special after-care. The Penrose tube is removed on the third day, and the sutures shortly after. Any intercostal water seal is removed when the lung is fully re-expanded and the lung fistula closed.

**Results and Conclusions.** In 1955, Rydell and Jennings (19) reported three cases with severe degrees of mediastinal and subcutaneous emphysema. One arose from fractured ribs, the second from ruptured alveoli due to excessive straining while coughing, and the third from spontaneous pneumothorax due to ruptured emphysematous blebs. All three patients were dramatically relieved by emergency cervical mediastinotomy.

The operation is simple and is completed in a few minutes under local anesthesia,

## REFERENCES

- 1 Gaudiani, V The surgical treatment of suppurations in the posterior mediastinum, *Ann. Surg.*, 63 523 1916
- 2 Lürman, Ein Fall von Oesophagus-fistel mit secundärer Bildung eines Mediastinalabscesses, *Berl. klin. Wchnschr.* 1876 (Quoted in ref 1)
- 3 Nasslow Versuche über die Resection der Speiseröhre, *Arch. f. klin. Chir.* 54 1886 (Quoted in ref 1)
- 4 Neuhof, H. Acute infections of the mediastinum with special reference to mediastinal suppuration, *J Thoracic Surg.* 6 184 1936.
- 5 Adams, R. Acute suppurative mediastinitis, *J Thoracic Surg.*, 15 336 1946.
- 6 Burnett, W E. Recognition and management of mediastinitis, *Am. J Surg.*, 54 99 1941
- 7 Bell, J W., Beakin C. A., and Starkey G W B Mediastinal abscess following instrumental perforation of the esophagus, *Am. J Surg.*, 91 999 1956.
- 8 Macklin, M T., and Macklin, C. C. Malignant interstitial emphysema of the lungs and mediastinum as an important occult complication in many respiratory diseases and other conditions, *Medicine*, 23 281 1944
- 9 Fagin, I. D., and Schwab E. H. Spontaneous interstitial emphysema in pulmonary tuberculosis, *Ann. Int. Med.* 24 1052, 1946.
- 10 Karns, J R., and Dave, E. O Mediastinotomy in spontaneous mediastinal emphysema, *J.A.M.A.* 136 622, 1948
- 11 Iglauer S. Spontaneous mediastinal emphysema, *Ann. Otol., Rhin. & Laryng.*, 53 823 1944
- 12 Van der Laan, W P and Mareah, G The significance of mediastinal emphysema, *New England J Med.*, 235 617 1946.
- 13 Hammond, A. E. Emergency cervical mediastinotomy in a case of massive mediastinal and subcutaneous emphysema secondary to removal of a foreign body from the bronchus, *Ann Otol., Rhin. & Laryng.*, 53 829 1944
- 14 Scannell, J G Rupture of the bronchus following closed injury to the chest, *Am Surg.*, 133 127 1951
- 15 Burford, C. G The entrance of air into the mediastinum during operation on the base of the neck, *Surg., Gynec. & Obst.*, 26 540 1918
- 16 Forbes, G B., Salmon, G., and Herweg, J C. Further observations on post tracheotomy mediastinal emphysema and pneumothorax, *J Pediat.*, 31 172, 1947
- 17 Jessup, P M Mediastinal emphysema, *Arch. Surg.*, 20:318 1930
- 18 Hamman, L. Mediastinal emphysema, *J.A.M.A.* 128 1 1945
- 19 Rydell, J R., and Jennings, W B Emergency cervical mediastinotomy for massive mediastinal emphysema, *Arch. Surg.*, 70 647 1955

# Diaphragmatic Emergencies

## 21

### DIAPHRAGMATIC HERNIA

#### CONGENITAL DIAPHRAGMATIC HERNIA

In reviewing this not uncommon defect of the newborn, Hedblom (1) in 1925 found that the frequently conservative approach led to a mortality of 75 per cent before the end of the first month of life. Since then, it has been recognized that the earlier the patient is treated surgically the better, and that by this means from 90 to 95 per cent of these infants can be effectively and permanently cured (2)

**Embryology.** In the developing embryo, the abdominal and thoracic cavities are at first in free communication. Anteriorly, the heart becomes separated from the abdominal viscera by the developing septum transversum which forms the ventral half of the diaphragm. Posteriorly, proliferating mesodermal cells from the dorsal mesentery create a central bridge across the coelomic cavity but leave two lateral openings in the diaphragm—the *pleuroperitoneal canals*. These canals are later closed by the two serous layers of the developing pleura and peritoneum which finally become separated from each other and strengthened by the ingrowth of striated muscle. With the diaphragm derived from so many different sources, it is not surprising that developmental errors occur.

If development ceases before the serous layer forms, any subsequent hernia has no enveloping sac, whereas if it occurs *after* formation of the serous layer but *before* the appearance of striated muscle, a thin sac covers the hernial contents. In either case, adhesions between the pleural surfaces and the abdominal viscera are almost unknown.

#### PATHOLOGY

Congenital diaphragmatic hernia occurs most commonly

- 1 Posterolaterally, through either pleuroperitoneal canal (foramen of Bochdalek);
- 2 Anteriorly, through the foramen of Morgagni,
3. Posteriorly, through the esophageal hiatus; or, rarely,
- 4 Behind the aortic hiatus

Gross (2) in 1953 reported the following frequency

Left posterolateral	69 cases	Esophageal hiatus	5 cases
Right posterolateral	13 cases	Retrosternal area	4 cases

**Posterolateral Hernia.** This is five times as common on the left as on the right side, and in 9 of 10 of these hernias there is no sac. On the left side, the hernial contents include small and large intestine, a portion of the stomach and spleen, and the left lobe of the liver. On the right, there is usually small and large bowel, and liver which may be completely rotated so that its ventral surface with the gallbladder

lies uppermost in the chest. The gut may be malrotated. In an appreciable proportion of cases the abdominal cavity is relatively reduced in size.

Apart from a tendency to strangulate once the child starts to feed these hernias also severely restrict respiratory function, for the underlying lung is collapsed, the heart displaced, the normal lung compressed, and its ventilatory capacity greatly reduced.

**Anterior Hernias** Occurring through the foramen of Morgagni these are not nearly so extensive as the posterolateral type. Their progress is limited by the pericardium above and behind, and by the pleura on either side and they do not seriously affect respiratory function. Rarely herniation extends into the pericardial cavity. In 50 per cent of cases the hernia has a sac which may contain small or large bowel, or a portion of the stomach or liver.

**Hiatus Hernia.** Separation of the crurae of the diaphragm allows a sliding hernia of the bare area of the stomach up into the posterior mediastinum. There is always a hernial sac containing the stomach and rarely loops of intestine. This hernia allows reflux of gastric juice into the esophagus, and early onset of reflux esophagitis which, if uncorrected, can cause intractable esophageal stenosis. The author has seen this develop within six months (3).

### CLINICAL FEATURES

**Signs and Symptoms.** Although some hernias are symptomless, the majority produce alimentary respiratory and circulatory disturbances. With the young, clinical diagnosis rests solely on physical signs. Repeated vomiting, dyspnea or cyanosis in a newborn strongly suggest the diagnosis, especially of a posterolateral hernia.

**POSTEROLATERAL HERNIAS** These when large show diminished chest movement on the affected side, a heart displaced away from that side, a variable percussion note and absence of breath sounds. Rarely peristaltic sounds are audible. The abdomen is scaphoid and dull to percussion.

**HIATAL HERNIAS** At birth, a child with hiatal hernia may regurgitate most feedings, and lose weight rapidly. The author had a child in whom the diagnosis was made a week after birth because of persistent regurgitation.

If a stricture forms this causes dysphagia which is usually first detected when the child is weaned. Occasionally too the stricture becomes plugged by food causing total dysphagia. There are no typical physical signs.

**ANTERIOR HERNIAS** These rarely present as an emergency. They may remain symptomless for years or give vague symptoms of intermittent obstruction and show no typical physical signs.

**Investigations.** **ROENTGENOGRAPHY** This usually establishes the diagnosis but in addition, with hiatal hernia, esophagoscopy is important.

With a posterolateral hernia the plain film shows gut partly or completely filling the pleural cavity displacing the heart, and compressing the lungs (*Fig 1 left*). Gross (2) advised that it is wrong to give these newborn babies barium as it adds nothing to the diagnosis or treatment and may precipitate intestinal obstruction. An anterior hernia may show a ventral shadow on one side of the heart or the other. At times it can be differentiated from a pericardial cyst only by giving barium by mouth and following it through the alimentary canal.

With a hiatal hernia, examination in the Trendelenburg position w

the sliding hernia and incompetent cardia that allow gastric reflux into the esophagus (Fig 1)

**ESOPHAGOSCOPY.** This will confirm the presence of reflux and will show esophagitis, and it may be necessary to remove retained food if a stricture has already developed

### TREATMENT

Experience has led to the conclusion that the only satisfactory treatment of these patients is early operation. The newborn child is far more fit to withstand operation than a baby who is allowed to wait a week or 10 days in the hope that he will become stronger. Also, the operation on the newborn is technically easier as the bowel is not yet distended and can readily be replaced into the abdominal cavity.

**Preoperative Treatment.** The baby requires adequate hydration, decompression of the alimentary canal by a stomach tube and enemas, and oxygen therapy. The stomach tube remains in position during the operation as a safeguard against accumulation of gastric fluid and as a means of enhancing deflation of the bowel.

Blood transfusion may be given into the umbilical vein in the newborn or into a saphenous vein in an older infant.

**Anesthesia.** An endotracheal tube is passed and the child lightly anesthetized with trilene, or with gas, oxygen, and ether, according to preference. In either case, diathermy cannot be used.

**Repair of Posterolateral Hernias. APPROACH** While Gross advocates an abdominal approach for babies and has found it allows simultaneous correction of any malrotation of the gut, the author has experienced no difficulty in reducing such a hernia through a thoracic incision. Especially, when the hernia is right-sided and with the liver also in the pleural cavity, he feels the thoracic incision gives easier access to the neck of the hernia and to the displaced organs and so makes repair of the hernial defect much easier.

**REDUCTION OF THE HERNIA.** With the baby anesthetized and turned on his side, the chest wall is opened through an eighth or ninth intercostal space incision. The neck of the hernia is sought and the opening enlarged by incising the diaphragm laterally for 2 to 3 cm. The hernial contents are then easily reduced into the abdomen. If there is a true hernial sac, this is incised, reduction carried out as just described, and thereafter the sac is dissected free and removed at the level of the hernia orifice.

During a brief pause, the anesthetist inflates the underlying lung.

**REPAIR OF THE HERNIAL DEFECT** The edges of the hernial defect are lifted up with pairs of stay sutures. They are then approximated by a series of interrupted No. 4 silk mattress sutures, if possible overlapping the edges, and using two rows of sutures. If the defect is too large to approximate the edges, it may be bridged by using Tantalum wire mesh.

**CLOSURE OF THE CHEST WALL** Finally, the underlying lung is reinflated, and the chest wall is closed over water-seal drainage with interrupted sutures for the muscle and subcutaneous layers, and nylon for the skin.



Fig. 1A. Right-sided congenital diaphragmatic hernia.

**CASE REPORT** J. M. female, aged 11 months, was admitted on May 16, 1952. Her mother's pregnancy and confinement had been normal, and her birth weight was 7 lb 4 oz. At two months, she was weaned, but she commenced to have frequent vomiting, often several hours after feeding. Her mother sought medical advice because of the infant's increasing dyspnea and unilateral heaving of the chest. Roentgenograms confirmed a right congenital diaphragmatic hernia (Fig 1A).

This was repaired on May 21. The anesthetist gave gas, oxygen, and ether through an endotracheal tube, and an intravenous drip was started in the right internal saphenous vein. The right pleural cavity was opened through the ninth intercostal space. There was no hernial sac. The pleural cavity contained the whole of the small intestine and the greater part of the large intestine as well as the liver. The hernial orifice was enlarged by a 2 cm lateral incision. The

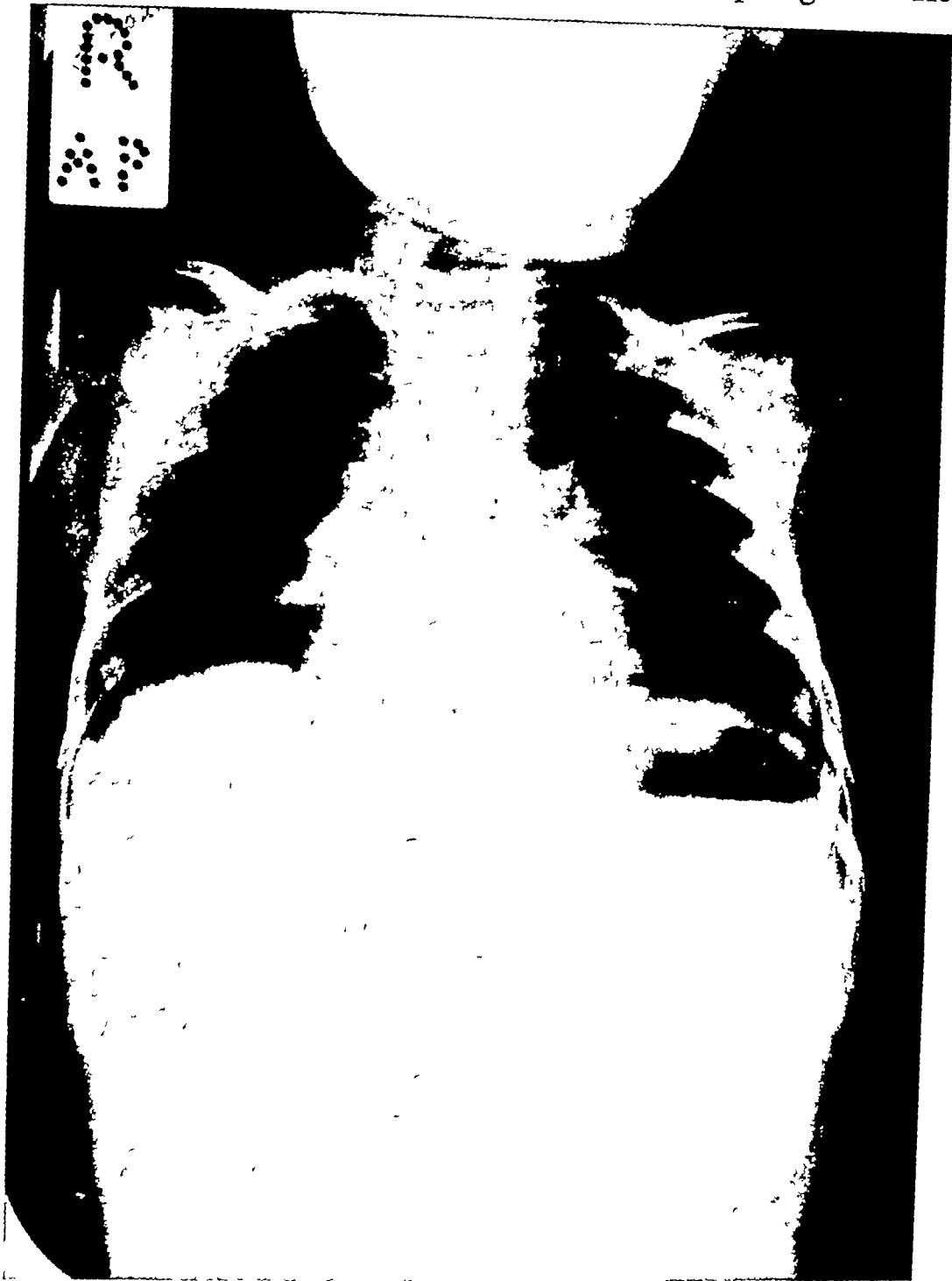


Fig 1B Film one month after transthoracic repair

the intestine was easily reduced and revealed that the liver was completely in the pleural cavity, having rotated on its ligaments so that the ventral surface was facing the apex of the right pleural cavity. After dividing a few adhesions between diaphragm and liver, the liver was reduced into the abdominal cavity. The preoperative fear that there would not be enough room in the abdomen for the herniated viscera proved unfounded.

The right lung immediately re-expanded to fill three quarters of the right pleural cavity. The diaphragm was then radially repaired in two layers with interrupted strong silk sutures. There appeared to be no loss of diaphragmatic substance. The chest cavity was closed with water-seal drainage, fine silk sutures being used for all deep layers and nylon for the skin. At the end of the operation, the water-seal drain was removed. The baby was nursed for the next day in an oxygen tent. Her convalescence was uneventful.



Fig. 2. Hiatal hernia in child, aged one week.

*Postoperative Care* Intravenous therapy continued for 24 hours. Chest films confirmed that the lung had re-expanded. Feeding by mouth was started on the second postoperative day; bowel sounds were audible on the third day and her bowels functioned on the fifth day after operation. A week later she was discharged (Fig. 1B).

**Anterior Hernia.** This is similarly approached from the chest and similarly repaired. The author has also used a midepigastrie approach with success.

**Hiatal Hernia** (Fig. 2). The urgency for operation for hiatal hernia is twofold:

1. There may be a failure of the infant to thrive with rapid decline in health.
2. There is the ever present danger of stricture formation.

The technic of operative repair differs little from that used on adult patients. The left chest is opened through the eighth intercostal space. The pulmonary ligament is divided, the esophagus is isolated in the posterior mediastinum and a catheter sling is placed around it (Fig. 3 A). Next, a small opening is made in the left dome of the diaphragm, and a pair of O'Shaughnessy forceps is introduced and passed up through the esophageal hiatus into the hernial sac. The sac is then opened, the crura of the diaphragm dissected free (Fig. 3 B) and the hernia reduced by threading the catheter sling onto the O'Shaughnessy forceps and withdrawing them together (Fig. 3 C).

The crura of the diaphragm are next approximated behind the esophagus with three or four 4-0 silk sutures and the esophageal wall is further sewed to the diaphragm with interrupted fine silk sutures (Fig. 3 D). The diaphragmatic opening is closed with silk mattress sutures, and the chest wall is closed in layers over water-seal drainage.



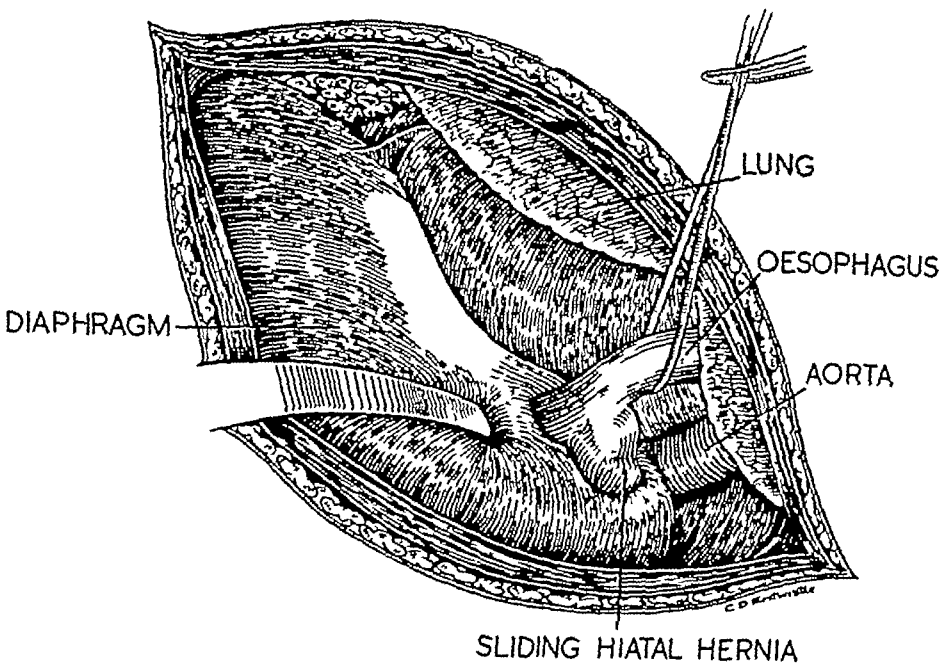


Fig 3A. Technic of repair of hiatal hernia Transthoracic exposure of hiatal hernia.

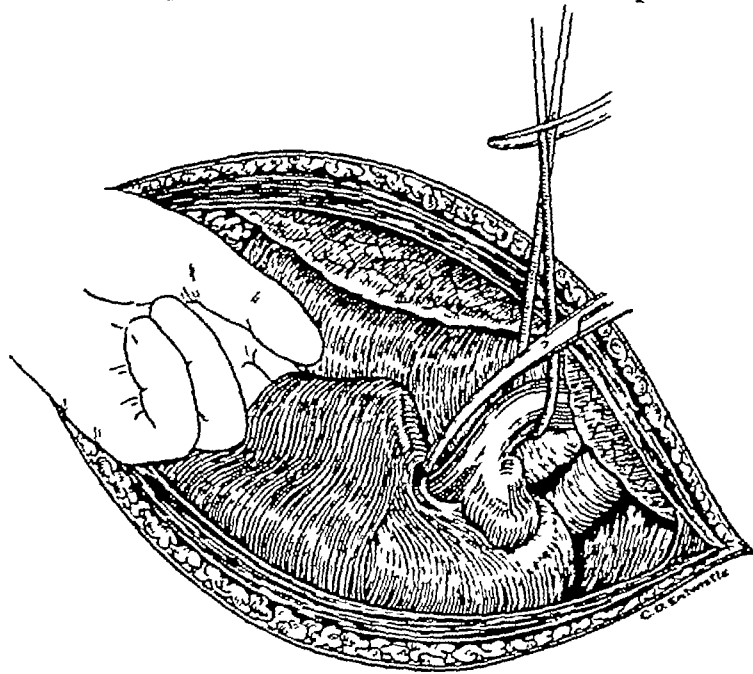


Fig 3B Technic of repair of hiatal hernia Catheter sling for retraction has been passed around the esophagus, small incision made in the left diaphragm, and the hernial sac incised

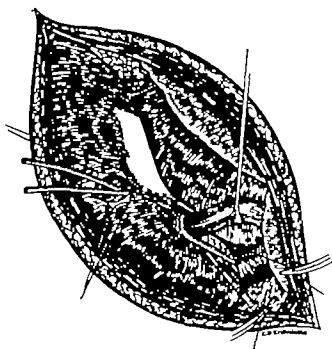


Fig. 3C. Technic of repair of hiatal hernia. The hernia is reduced by passing the catheter ends through the hernial orifice and out through the diaphragmatic incision. Interrupted strong silk sutures have been placed between the crura of the diaphragm behind the esophagus.

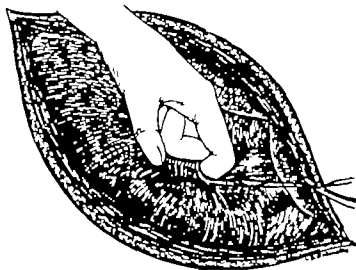


Fig. 3D. Technic of repair of hiatal hernia. The interrupted sutures are tied, the diaphragmatic opening closed, and the esophagus anchored to the diaphragm with interrupted silk sutures. (Figs. 3A-D from Borrie, J., in Malngot, R., *Management of Abdominal Operations*, 2nd Ed., 1957. Courtesy H. K. Lewis & Co. Ltd., London.)

**CASE REPORT.** A S, female, aged 1 week, was admitted to the pediatric department, on September 3, 1953, because of persistent vomiting. The birth weight was 6 lb 10 oz, but on admission she weighed only 5 lb 9½ oz. Lipiodol study of the stomach revealed a hiatal hernia. She was at first treated medically and nursed in the vertical position supported in a plaster cast. As her weight, however, after six weeks had risen only to 6 lb. 15 oz. and as she was developing pressure sores and was not thriving, she was referred for operation.

The left pleural cavity was opened through the left eighth intercostal space. There were no adhesions. The inferior pulmonary ligament was divided. With each expiration, the stomach slid well up into the posterior mediastinum, causing a considerable bulge behind the heart. The mediastinal pleura was incised vertically and a finger passed round the esophagus. No fibrous stricture was detected. The hernia was reduced and repaired as described above, and the chest wall was closed over water-seal drainage which was removed at the end of the operation after the lung was fully reinflated.

At the age of two years, she had a further episode of regurgitation suggesting too lax a closure of the cardia. The regurgitation was treated medically, however, and ceased within a month. At the age of four years, she was fit and of normal weight for her age.

**Results.** Gross reported that, in an 11-year period, of 72 cases, 3 died from other causes, 5 from the hernia or 1 of its complications, and 64 patients (89 per cent) were cured of the anomaly. Size of the child is no bar; Clinton-Thomas (4) reports successful repair of a left posterolateral hernia via the abdomen in a premature babe weighing 4 lb 8 oz. on the third postnatal day.

### CONCLUSION

Operation should always be advised.

1. To relieve symptoms,
2. To avoid such complications as stricture formation with a hiatal hernia or strangulation if the hernia is posterolateral. These complications are hazardous in the extreme to manage and are best avoided.

### TRAUMATIC DIAPHRAGMATIC HERNIA

**Introduction.** Traumatic diaphragmatic hernia is rightly regarded as an emergency (5, 6), if only because of its serious possible complications—obstruction, strangulation, and perforation—and because 90 per cent of strangulated diaphragmatic hernias are traumatic in origin.

### PATHOLOGY

The most common *causes*, especially in wartime, are penetrating stab, gunshot, or shell-fragment wounds. Closed trauma, from crushing of the chest or abdomen in automobile accidents or falls from heights (8), is next most common, while post-operatively, the hernia may arise after incision of the diaphragm.

In hernias due to crush injuries, as Evans and Simpson (9) suggest, there is an explosive rupture across the dome of the diaphragm from a sudden increase of intra-abdominal pressure. In a series of 28 cases, Hughes and associates (10) found 17 due to penetration and 11 due to rupture of the diaphragm. Regarding site, because of the splinting action of the diaphragm, the right side is less commonly affected. Al-

though Hedblom (11) believed 95 per cent were left sided, Hughes reported 5 on the right side and 23 on the left. Rarely traumatic hernias involve the esophageal hiatus but often they extend from it forward across the dome of the diaphragm. At times, the diaphragm may be torn from its costal attachment.

The hernia need not necessarily be obvious immediately after injury but may develop later as occurred in 5 of Hughes cases. One in fact, herniated 13 months after injury. In Hamdi and Sturdy's (12) case, the herniation occurred from the patient's straining while under anesthesia for suture of other traumatic injuries. The traumatic rent in the diaphragm tends to be kept open (a) by the negative pressure of the chest, causing ascent of abdominal organs through the new orifice and (b) by omentum separating the two edges (13). Associated lesions may also occur such as fractured pelvis, which Evans and Simpson found in four of seven cases.

### CLINICAL FEATURES

**Symptoms.** As Carter (13) points out, these fall into three groups. *The hernia may be discovered*

- 1 At the time of injury
- 2 After a latent interval when it subsequently develops and causes symptoms
- 3 Finally when complications such as strangulation occur

At the time of injury the patient may be shocked and dyspneic and may have an external wound or multiple rib fractures with pneumothorax. These often mask any specific symptoms of the diaphragmatic hernia which is often recognized only after roentgenography. Symptoms when present, are essentially those of disturbed cardiorespiratory function and at times of intestinal obstruction. Pain, dyspnea, tachycardia, hypotension, mediastinal shift, and reduced ventilatory function all result from collapse of the underlying lung. If intestinal obstruction occurs, these signs are added. When the hernia develops later there is usually a history of straining or lifting which precipitates the above-mentioned symptoms.

When the hernia is not recognized at the time of injury, symptoms tend to vary with the nature of the hernial contents. Herniation of liver or spleen can be associated with vague lower chest pain. Herniation of stomach or intestine may produce thoracic symptoms such as dyspnea or unusual chest gurgling, or abdominal symptoms such as nausea, flatulent dyspepsia, epigastric pain, and vomiting, especially after meals or when recumbent. The severity of symptoms depends on the degree of herniation and intestinal obstruction (see below).

**Physical Signs.** The distinctive physical signs listed by Gibson (14) in 1929 still apply

- 1 Diminished excursion of the thorax
- 2 Impaired resonance
- 3 Absence of Litten's diaphragm phenomenon
- 4 Changeable adventitious sounds in the chest
- 5 Cardiac displacement
- 6 Circulatory collapse

**Diagnosis.** The possibility of traumatic hernia lesion should always be kept in mind and the pathway of any missile reconstructed to see if a hernia could have occurred. Careful roentgenography as emphasized, is essential in clinching the diagnosis.

While large hernias may be obvious because of an unusually high diaphragm, abnormal densities, air pockets, areas of atelectasis in the subjacent lung, and mediastinal shift to the right, lesser degrees are more puzzling. When a left leaf of diaphragm is higher than the right, with a history of trauma, the diagnosis of hernia should be entertained until proved otherwise. Confirmation of the diagnosis may require the passing of a Levin tube to the stomach, or, if nonacute, a barium meal or barium enema to delineate the hernial contents and the degree of obstruction. Unger (15) reported a right-sided traumatic diaphragmatic hernia which had clinical and x-ray appearances simulating a pleural effusion.

When a chest roentgenogram is "negative," especially after a stab wound, serial films are required to detect a late herniation.

### TREATMENT

Treatment is by surgical repair of the defect. The transthoracic approach gives a better exposure than is possible through the abdomen, allows safe division of adhesions, and facilitates closure of the defect. If necessary, it can be extended across the left costal margin as a thoracoabdominal incision.

**Preoperative Care.** The general measures of pre- and postoperative care outlined in Chapter 3 apply. Whether there is any degree of intestinal obstruction or not, a Levin tube is inserted through the nose to the stomach, and the stomach contents are aspirated before and during the operation, especially at the time of reduction. The tube should remain in place for a time after operation. When the colon has also herniated, a rectal tube will assist deflation.

**Operative Treatment.** Left thoracotomy is performed through the seventh rib bed. When the pleural cavity is exposed, hernial contents are seen. Adhesions between the hernial contents and the chest wall are divided. The edges of the diaphragmatic rent are lifted up with stay sutures or Allis tissue forceps, and herniated viscera are returned to the abdominal cavity. The diaphragmatic defect is then closed either with two layers of interrupted silk mattress sutures or by imbricating the rent. When there is a considerable defect with tension after the repair, either phrenic nerve paralysis or the use of tantalum mesh should be considered.

If there is a flail chest wall, it is stabilized by the technic illustrated in Chapter 5. The chest wall is finally closed over water-seal drainage.

**Postoperative Care** (see Chapter 3). If there is any degree of intestinal obstruction, intravenous therapy is continued until normal bowel function returns. The gastric tube is removed when the aspirate is clear and normal in amount.

**Results.** In their 28 cases Hughes and associates (10) reported that obstructive symptoms necessitated emergency operation in 5, and the diagnosis was not made until thoracotomy in 8. All were successfully repaired by the transthoracic approach.

Carter and associates (13) reported 2 deaths in 11 cases, both associated with strangulation of the bowel. One was a postoperative hernia.

### CONCLUSION

The train of events after diaphragmatic injury is unpredictable. The severity of the immediate lesion varies. Hours or months may elapse before signs of hernia appear. Complications greatly increase the mortality rate, and prompt surgical repair gives the patient his best chance of recovery.

## STRANGULATED DIAPHRAGMATIC HERNIA

**Introduction.** Strangulation is the most serious complication of a diaphragmatic hernia. This condition was first described in 1575 by Paré (17) Prior to the use of roentgenography strangulation was seldom diagnosed

### PATHOLOGY

Of 43 cases of hernia with strangulation, Carter and Guiseffi (14) found only 4 were congenital while 34 were traumatic in origin

Age offers no security for strangulated hernias have been reported in babies 2, 6 and 12 weeks old (18, 19 20), as well as in adults aged 65 and 74 years (21, 22) In the author's 3 cases summarized here the ages were 11, 22 and 30 years and were respectively congenital, traumatic, and hiatal in origin.

The hernial ring is usually an opening in the dome of the diaphragm or rarely in a foramen of Morgagni Strangulation of a hiatal hernia also occurs and can be associated with torsion of the stomach on its long axis (23)

Though small and large bowel strangulate more readily than the stomach, the latter is not immune (24) nor is strangulation confined to the left side only The distended bowel causes acute displacement of the mediastinum to the contralateral side with gross limitation of ventilatory function. This is aggravated by an outpouring of blood-stained pleural effusion, which Carter and Guiseffi (7) found in 30 per cent of cases

### CLINICAL FEATURES

**Signs and Symptoms.** In a male patient, there may be a history of recent or of remote chest trauma as well as a variable period of indefinite symptoms, ending in an acute exacerbation with intestinal obstruction and respiratory distress, sometimes starting after a festive meal

A typical case presents a triad of agonizing, griping, epigastric pain radiating up behind the sternum, accompanied by severe *dyspnea* and by *vomiting* that rapidly becomes feculent in character In some patients blood and mucus may be passed by rectum in others there is constipation.

Unless the practitioner has the possibility of strangulated hernia in mind, the complex nature of the physical signs can at first sight obscure the diagnosis as in the following case report (25)

**CASE REPORT** A soldier aged 22 years, was seen by the author in a German prisoner-of-war working camp on December 26, 1942, 7 hours after onset of symptoms. There was cyanosis of the face and extremities, short gasping respirations at 50 per minute, a temperature of 101.2 F., and a weak irregular pulse rate of 120 per minute. To ease distress, the patient insisted on lying on his left side, and he was too ill to give a coherent history

Abdominal examination showed an acutely tender and rigid epigastrium without bowel sounds. In the chest the trachea was displaced to the right sternal border the heart had a maximal impulse in the right fifth rib space 9 cm. from the mid sternal line, and heart sounds were feeble, fading, and irregular

Chest expansion was limited on the left there were a palpable inspiratory and expiratory fremitus, alternating areas of dullness and hyperresonance, bronchial breathing, whispering pectoriloquy and no egophony



Fig 4 Roentgenogram of strangulated left congenital hernia 40 hours after onset

An enema was returned unaltered. Chest aspiration of 500 ml of odorless, turbid, blood-stained fluid temporarily improved the clinical condition. Further examination then revealed a 2.5 cm axillary scar between the left eighth and ninth ribs, while a later change in physical signs and aspiration of 60 ml of feculent fluid confirmed a belated diagnosis of strangulated diaphragmatic hernia.

**Diagnosis.** ROENTGENOGRAMS These were not available for the patient just described. When they are available, they provide the short cut to diagnosis. The herniated viscus usually simulates a high diaphragm and may show uniform opacity of a hemithorax or a grossly distended stomach or coils of bowel and pleural fluid. Almost invariably, the mediastinum is displaced to the opposite side (Figs 4 and 5).

*Diagnosis, therefore, is suggested by*

1. Signs of acute gastrointestinal obstruction,
2. History of an old injury,
3. Physical signs in the left thorax,
4. Roentgenologic evidence of a high left diaphragm.

**Mortality.** Because of the remoteness of the lesion, the vagueness of early symptoms, and the complexity of signs resulting in delay of final diagnosis, mortality has always been high. Before 1920, it was over 88.8 per cent, with an operative mortality of 66 per cent, while in the period 1920 to 1945, Carter and Guiseffi (17) found a general mortality of 57.1 per cent and an operative rate of 43.7 per cent.

### TREATMENT

Operation is required. Because of pleural adhesions, a thoracic incision that can have an abdominal extension is advised, preferably through the eighth rib bed.

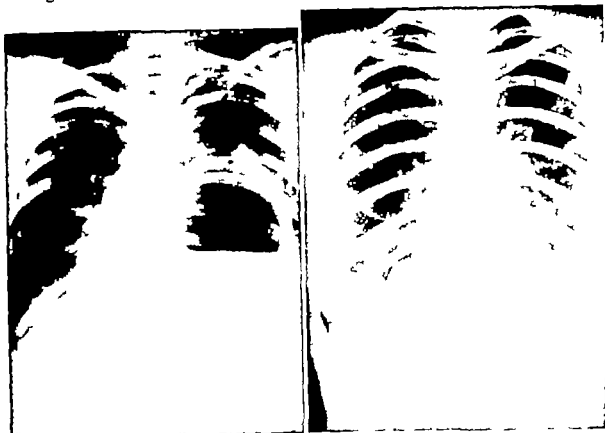


Fig. 5 Roentgenogram of strangulated hiatal hernia developing in pregnant woman at term. Right, same patient' roentgenogram of chest 18 months earlier (Courtesy R. Nicks and T. Plunkett.)

**Preoperative Preparation.** This follows the outline given in Chapter 2, with emphasis on passing a Levin tube into the stomach, giving an enema, and starting a blood drip.

**Operation.** Anesthesia should be light and any intubation rapidly performed, lest the release of muscle tone allow the tensely distended coils of gut to compress completely the remaining normal lung and so cause cardiac arrest.

The details of the operation differ little from those for traumatic diaphragmatic hernia. Any fluid is sucked away. The constricting neck of the hernia is sought and incised laterally with immediate relief (Fig. 6A). If the bowel recovers it is then replaced in the abdomen, the diaphragm is repaired as described above, and the chest wall closed (Fig. 6B). When there is necrosis, bowel resection is performed along standard lines.

The following two case reports illustrate the problems of surgical relief.

**CASE REPORT D P.** a Maori girl, aged 12, was admitted under the author's care on April 13, 1952, with a 40-hour history of strangulated left congenital diaphragmatic hernia through the foramen of Bochdalek. The hernia had been diagnosed at the age of two, but treatment had been refused. Roentgenograms confirmed the diagnosis of strangulation.

A stomach tube was passed and brown gastric contents withdrawn. Anesthesia was induced with thiopentone, and during intubation the patient suffered cardiac arrest. Because of the strangulated hernia, a left thoracotomy approach was out of the question and a midline epigastric one was chosen. One ml. of 1/1000 adrenalin hydrochloride was injected into the heart and effective cardiac compression obtained between diaphragm and sternum with immediate return of a forceful heart beat.



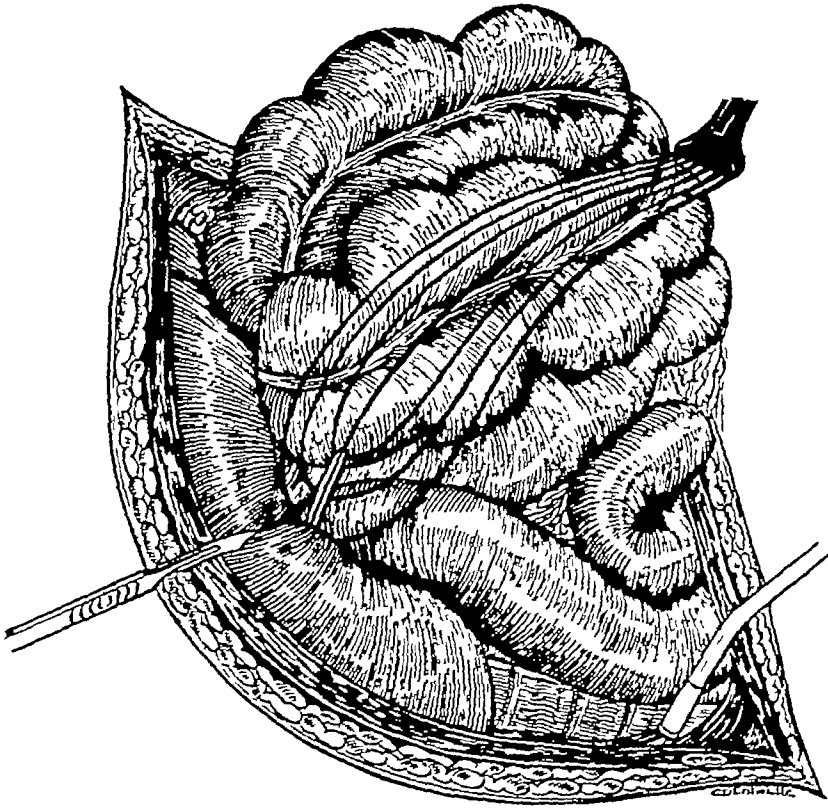


Fig. 6A Technic for repair of left strangulated diaphragmatic hernia via left thoracotomy  
Lateral incision in hernial ring is valuable especially where hernia is incarcerated

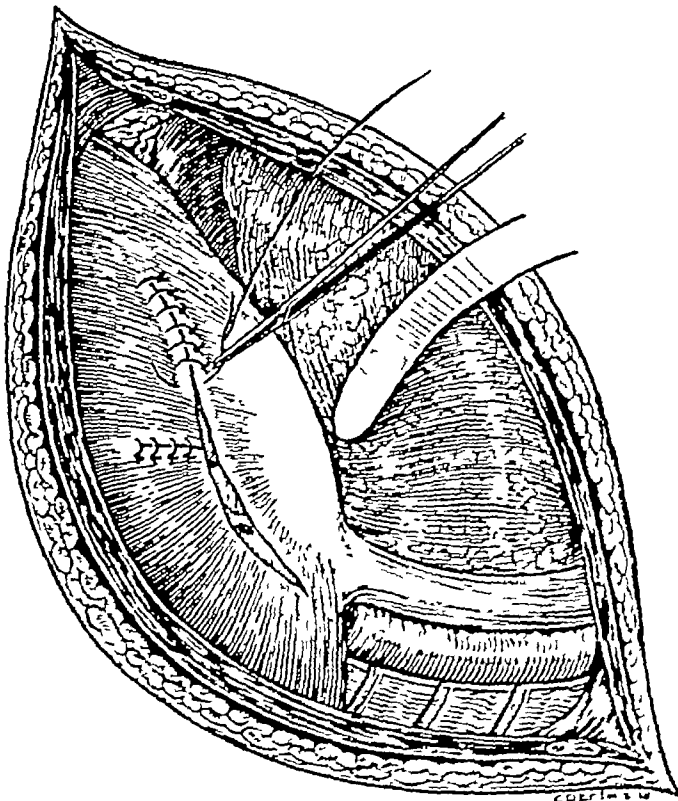


Fig 6B Repair of hernia with interrupted silk sutures

Left thoracotomy then revealed strangulation of the stomach spleen greater and lesser omenta, the whole of the small bowel, and the greater part of the large bowel. Adhesions were divided and the neck of the hernia was enlarged by a lateral incision with immediate recovery of the bowel. The spleen, stomach, large bowel, omenta, and small bowel were reduced in turn, without difficulty and the hernia repaired. Both the thoracotomy and abdominal incisions were closed.

The patient was slow to regain spontaneous respiration. She remained in coma and died as the result of cerebral anoxia from her episode of cardiac arrest eight hours after operation.

*Strangulated hernia has no respect for time or place*

**CASE REPORT** Mrs S H aged 30 was admitted to Cornwall Hospital Auckland, on December 11 1951 complaining of abdominal pain and nausea. She was pregnant and one week from term. Her earlier chest film had been normal. Admission roentgenograms showed a large diaphragmatic hernia completely filling the left pleural cavity. The stomach was grossly distended with fluid and air. There was also large bowel present. Clinically the patient was suffering from a strangulated diaphragmatic hernia. (Fig 5)

Caesarean section was performed, and the child was delivered dead. The abdominal incision was enlarged to the xiphisternum and the hernia found passing through a large hole in the back of the diaphragm. Even after incising the hernial ring, the hernia could not be reduced. Some fluid and air however could be expressed from above downward. Thereafter with gentle traction for 10 minutes, the hernia was reduced. It contained cyanotic but viable stomach large bowel and small bowel which quickly recovered. After reduction, the abdominal contents were packed away the diaphragmatic opening was visualized lying in the hiatal region. It was closed with interrupted silk sutures. An intercostal water-seal drain was inserted into the left chest, and the abdomen was closed with silk sutures. Postoperative ileus was treated with gastric suction and intravenous therapy for the following week. The patient's left lung completely re-expanded, and she made a satisfactory recovery (26).

**CASE REPORT** W G., a baby girl nine months old was an urgent admission to Wakari Hospital Dunedin on May 18 1958. She had coughed for a week, vomited fluids for four days, and cried when moved. She was severely dehydrated, with depressed fontanelles. Temperature 100° F pulse 160 per minute, and respirations 40 per minute. Besides signs of bronchopneumonia, there was a striking bulging of the precordium hyperresonant to percussion. X ray films, which showed a large air-containing "cyst" completely filling the anterior mediastinum and displacing the heart backward, confirmed the diagnosis of *strangulated anterior diaphragmatic hernia*.

After rehydration by subcutaneous and intravenous therapy the baby was anesthetized, and a midline epigastric incision made. An obstructed loop of transverse colon was reduced, followed by half the liver. The sac reached up to the sternal angle. The hernial orifice was repaired by sewing the diaphragm to the costal and sternal margins. The wound was closed in layers. Despite temporary ileus, treated by gastric suction, her recovery was complete.

**Postoperative Treatment.** The general measures outlined in Chapter 3 apply. In addition, attention must also be paid to the ileus accompanying relief of the obstruction. A Levin tube is left in the stomach until aspirations are clear and bowel sounds audible. Intravenous therapy continues until the bowels move and fluid balance is accurately charted.

## CONCLUSION

Prompt correction of congenital and traumatic hernias prevents strangulation with its greater risks and difficulties. If strangulation develops, however, treatment aims at relieving the intestinal obstruction and repairing the hernia at the one operation, usually via a thoracic approach.

## REFERENCES

- 1 Hedblom, E C Diaphragmatic hernia. A study of 378 cases in which operation was performed, *J A M A*, 85 947, 1925
- 2 Gross, R E *The Surgery of Infancy and Childhood*, Philadelphia, W B Saunders Co, 1953
- 3 Borrie, J In Maingot's *The Management of Abdominal Operations*, 2nd ed, London, H K Lewis & Co, Ltd, 1957
- 4 Clinton-Thomas, C L Congenital diaphragmatic hernia in a premature infant, *Lancet*, 1 1155, 1955
- 5 Lam, C R Treatment of traumatic hernia of the diaphragm, *Arch. Surg*, 60 427, 1950
- 6 Bugden, W F, Chu, P T, and Delmonico, J E. Traumatic diaphragmatic hernia, *Ann Surg*, 142 851, 1955
- 7 Carter, B N, and Giuseffi, J Strangulated diaphragmatic hernia, *Ann Surg*, 128 210, 1948
- 8 Cooper, A *The Anatomy and Surgical Treatment of Hernia*, Philadelphia, Lea and Branchard, 1844 (American edition of work originally published in 1804)
- 9 Evans, C J, and Simpson, J A Fifty-seven cases of diaphragmatic hernia and eventration, *Thorax*, 5 343, 1950
- 10 Hughes, F, Kay, E B, Meade, R H, Hudson, T R, and Johnson, J Traumatic diaphragmatic hernia, *J Thoracic Surg*, 17 99, 1948
- 11 Hedblom, C A Diaphragmatic hernia, *Ann Int Med*, 8 156, 1934
- 12 Hamdi, F A, and Sturdy, D E Diaphragmatic hernia following injury, *Lancet*, 1 1001, 1955
13. Carter, B N, Giuseffi, J, and Felson, B Traumatic diaphragmatic hernia, *Am J Roentgenol*, 65 56, 1951
- 14 Gibson, F S The diagnosis of diaphragmatic hernia with acute obstruction, *J A M A*, 93 1719, 1929
- 15 Unger, S M Right-sided traumatic diaphragmatic hernia simulating a pleural effusion, *J A M A*, 151 734, 1953
- 16 Paré, A *Les Oeuvres*, Paris, G Buon, 1575
- 17 Carter, B N, and Giuseffi, J Strangulated diaphragmatic hernia, *Ann Surg*, 128 210, 1948
- 18 Rickham, P P Strangulated diaphragmatic hernia in neonatal period, *Thorax*, 10.104, 1955
- 19 Cooke, R V, and Corner, B Recurring left diaphragmatic hernia with intestinal obstruction by adhesions, *Proc Roy Soc Med*, 42 914, 1949
- 20 Ficarra, B J Incarcerated congenital diaphragmatic hernia, *Am J Surg*, 92 472, 1956
- 21 Watkins, D H, Harper, F R, and Condon, W B Diaphragmatic hernia with visceral complications, *Arch Surg*, 65 95, 1952
- 22 Markle, G B Strangulated right-sided diaphragmatic hernia, *Arch Surg*, 72 273, 1956
- 23 Sellors, T H, and Papp, C Strangulated diaphragmatic hernia with torsion of stomach, *Brit J Surg*, 43 289, 1955
- 24 Hamilton, J E, and Phillips, T W Traumatic hernia of the diaphragm with strangulation and gangrene of the stomach. Report of two cases, *Am J Surg*, 78 686, 1949.
25. Borrie, J, and Foreman, H M Strangulated traumatic diaphragmatic hernia, *Lancet*, 2:12, 1948
26. Plunkett, T, and Nicks, R Personal communication

### CARDIAC WOUNDS AND HEMOPERICARDIUM

Wounds of the heart constitute a grave emergency requiring prompt, informed handling if life is to be saved.

**Historical Note** The history of this ancient lesion is well described by Beck (1) Experimentally in 1882 Block first sutured cardiac wounds and in 1895, Del Vecchio demonstrated sutured wounds in dog hearts On September 9, 1896 Rehn of Frankfurt first successfully sutured the heart of a man aged 22 years which had a right ventricular wound 1.5 cm long Matas (2) in 1897 explored a nonpenetrating left ventricular wound that required no suture In 1934 Ramsdell (3) collected 428 surgical cases in which the mortality averaged 50 per cent

During the late thirties, with increased awareness of what surgery could do for such lesions, the numbers treated surgically steadily increased In 1943 Nelson (4) reported a series of 11 cardiorrhaphies with 1 death—a mortality of 9 per cent Nevertheless, with such an element of chance in these wounds, it is unlikely that a greater reduction of over-all mortality can be hoped for

#### PATHOLOGY

An indication of general statistics is seen from a series of 81 patients reported by Maynard and associates (5) Of these 20 died before operation could be done 61 were operated upon of whom 35 recovered In this series, 67 were males and 14 females Of these 69 were in the 20-to-40-year age group The youngest was 15 the oldest 61 Regarding causation of the injuries, 1 was a gunshot wound, 4 were made by an ice pick, and the remaining 76 were knife wounds

In general, while most cardiac wounds are penetrating wounds a few are the result of blunt trauma to the chest (6) The injury is common in war and not all of these casualties prove fatal Of 2,811 chest casualties that reached one hospital during the Korean War Valle (7) reported that 117 had injuries to the heart and mediastinum—an incidence of 4.2 per cent In this series 90 per cent were penetrating injuries with retention of foreign bodies and 10 per cent were perforating injuries

Pathologically these wounds fall into two groups *those of the heart alone* and *those complicated by associated injuries* of the other viscera, especially the lungs

**Wounds of the Heart Alone.** These fall into two broad groups

- 1 Wounds that cause early death
- 2 Wounds that allow the patient to reach a hospital

**WOUNDS THAT CAUSE EARLY DEATH** The wound may be large and prove fatal within a few seconds or it may be small and allow some activity as described by Davidson and Fiddes (8) whose patient, stabbed in the chest, walked 550 meters in 20 minutes before collapsing and dying Many more examples are cited by Gould and

Pyle (9) Rarely, hemopericardium may be delayed beyond six days and appear as a secondary hemorrhage (10).

**WOUNDS THAT ALLOW THE PATIENT TO REACH A HOSPITAL** The possible sites of such wounds and their wide variety are seen from Maynard's 61 cases that came to operation. In this series, 12 had pericardial wounds alone and 49 had myocardial wounds—3 in the right atrium, 3 in the left atrium, 20 in the right ventricle, and 23 in the left ventricle. In 7 patients, the coronary vessels were lacerated—1 case involved a small branch of the left coronary; 2 cases, the anterior descending branch of the left coronary, both near the apex, and 4 cases, branches of the right coronary. Three patients with wounds that penetrated both left and right ventricles from the anterior to the posterior surfaces of the heart all recovered.

**Cardiac Tamponade** This is the fundamental lesion in cardiac wounds that survive long enough to allow of treatment. It is due to the escape of blood from the heart into the pericardial cavity. With the patient lying on his back, the resulting blood clot usually forms posteriorly.

**Physiologically**, cardiac tamponade affects both the venous and arterial sides of the circulation. On the *venous side*, rising intrapericardial pressure will interfere with venous return to the heart until, when the pressure is greater than that in the cavae, cardiac arrest occurs. Both experimentally and in practice, this can be alleviated by rapid intravenous infusion. On the *arterial side*, from cardiac compression tamponade lowers cardiac output and reduces blood pressure, thereby reducing coronary arterial blood flow. The end result is hypoxia. Tamponade has been reported in a patient for as long as 10 hours without fatality (5).

Beck (1) found experimentally in dogs that the injection of 150 ml of fluid into the pericardial cavity over a period of 30 minutes was fatal. In stab wounds of the heart when the opening in the pericardium did not allow the escape of blood through it, 250 ml produced a fatal tamponade.

**Wounds Complicated by Associated Lesions.** The most common of these is perforation of the pleura, which Maynard found present in 85 per cent of his cases. In 72 per cent, there was either hemothorax or hemopneumothorax. Sucking wounds of the chest occurred in 11 per cent of his cases.

As these associated lesions further decrease vital capacity and aggravate hypoxia, the necessity for rapid assessment and correction is obvious. Rarely, bilateral penetration of the thorax or thoracoabdominal wounds, including laceration of the diaphragm, may occur, while associated lesions of the esophagus, spinal cord, head, neck, and extremities have also been recorded, all of these must be remembered when assessing these patients.

**Late Complications.** Constrictive pericarditis may develop from organization of the hemopericardium in similar fashion to fibrothorax after hemothorax. This may be fully established in 6 months (11) or as late as 18 years (12) and is more likely to follow aspiration therapy than surgical repair of the lesion with evacuation of the hemothorax.

### CLINICAL FEATURES

There are also two broad clinical groups of patients that closely follow the pathologic grouping. They are

1. Those with a fatal injury,
2. Those who reach the hospital.

Those patients reaching hospital may have injuries so mild that close observation is all that will be necessary. Usually however the injury and general condition of the patient may be moderate to serious, and, since the one condition may rapidly progress to the other the closest vigilance throughout by the medical staff is essential.

**History** This may be provided by the patient or by a witness. Knowledge of the time, the instrument used, and the axis of force are essential in assessing the extent and nature of the damage.

**Tamponade** is unlikely to develop if there is a large pericardial tear that allows blood to escape externally into the mediastinum or into the adjacent pleural cavity. Further in the presence of small wounds of the atria, ventricles, or arteries because clot can develop rapidly progressive tamponade need not always occur (13).

**Clinical Examination.** Attention is directed to

- 1 The general appearance of the patient and signs of life
2. The site and nature of the wound
- 3 The state of the heart and lungs

In general, these patients are shocked and dyspneic the pulse is rapid and thready and, depending on the blood volume the neck veins may or may not be distended.

The type of wound is noted, whether sucking or otherwise. On auscultation, the heart sounds are distant and faint and the blood pressure low and unobtainable with a correspondingly low pulse pressure.

Examination of the lungs may reveal signs of hemothorax or tension pneumothorax with deviation of the trachea and apex beat. The course of the missile should be checked in order to determine if any of the associated lesions already mentioned could have occurred.

**Diagnosis** The presence of a chest wound near the heart, with profound bleeding, low blood pressure, high venous pressure and muffled heart sounds all point to cardiac injury. Clinical signs may support an associated lesion such as hemopneumothorax. Roentgenograms will confirm the presence of an associated lesion such as hemopneumothorax, but, unless they are taken serially, they are unlikely to aid in the diagnosis of cardiac tamponade.

**Cause of Death.** Chamberlain (13) stresses that cardiac tamponade per se as the cause of death is difficult to confirm, and that the main causes are

- 1 Alteration in the coronary circulation
- 2 Hemorrhage
- 3 Changes in pulmonary physiology which ultimately cause anoxia.

## MANAGEMENT

Successful management depends on a combination of

- 1 Antishock and supportive therapy
- 2 Further special tests
- 3 Aspiration of the pericardial cavity
- 4 Cardiorrhaphy

**Antishock Therapy** For success, this depends on the first examining doctor clearly appreciating that his prime duty is to treat shock by correcting blood volume thereby increasing cardiac output and peripheral resistance. The patient is best

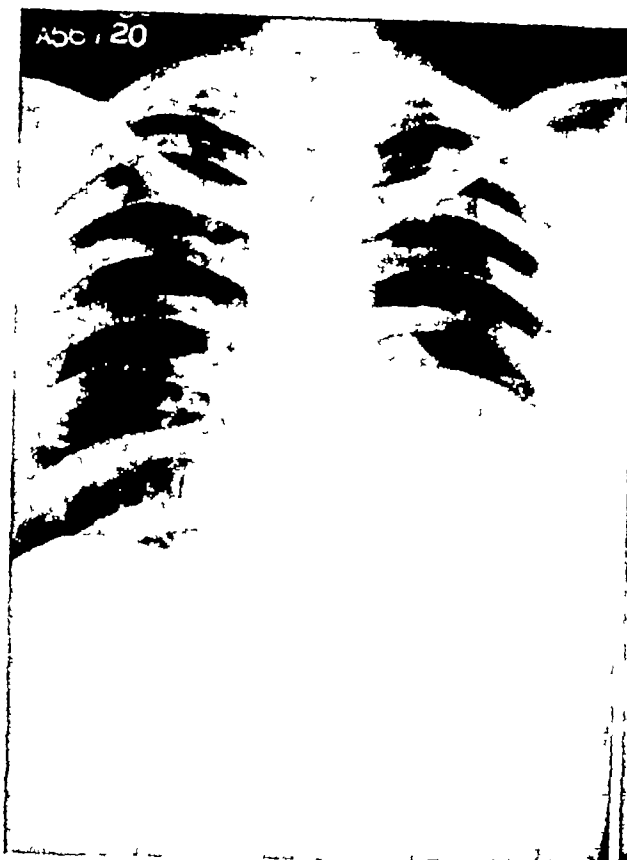


Fig 1A Roentgenogram of patient admitted with precordial stab wound with small hemopericardium



Fig 1B Roentgenogram of large hemopericardium

to a recovery room beside an operating room where all is made ready for emergency thoracotomy

The following steps are required:

- 1 Any sucking chest wound is firmly sealed with a pad and elastoplast, and pneumothorax is relieved by intercostal water-seal drain,
- 2 The airway is secured, and nasal oxygen is administered,
- 3 After a sample of blood for crossmatching is taken, an infusion of 5 per cent glucose in 0.25 per cent saline is started. This is changed to plasma and blood as soon as these are available,
- 4 Suitable sedatives are given for pain, and a special nurse is instructed to record pulse, blood pressure, and respiration rate at five-minute intervals. A surgeon must remain in constant attendance.

**Further Special Tests.** Griswold and Drye rightly point out that these can be carried out in only noncritical cases (14)

**ROENTGENOGRAPHY** A *fluoroscopic examination* of the heart is made at this stage and will be a guide to the degree of tamponade (Fig 1), while a *roentgenogram* taken with the patient sitting up will also show associated lesions, such as hemopneumothorax

**ELECTROCARDIOGRAPHY** This can be of value in verifying early the presence of pericardial and myocardial injury. The use of unipolar extremity leads and multipolar precordial leads is valuable in making the diagnosis of myocardial injury, even when the standard leads are normal

**Aspiration.** If tamponade is severe and there is no internal or external bleeding, pericardial aspiration is performed. With the patient lying on his back, if possible

with head and chest elevated, the epigastrium is infiltrated with local anesthetic and a 16 to 18 gauge needle inserted backward and upward to the left of the xiphoid process (Chapter 23 Fig 2) As soon as the pericardial cavity is entered up to 150 ml of dark blood may be aspirated. There is immediate relief, a fall in venous pressure a rise in arterial pressure, and return of a more audible heart beat. Farringer and Carr (16) reported improvement from aspiration of as little as 30 to 50 ml. of blood. If this occurs and the improvement is maintained, operation is correspondingly postponed. (If there is a slow relapse however further aspiration is undertaken as a prelude to operation. Sedatives are given four hourly as required.) When blood pressure and hemoglobin finally return to normal, the intravenous infusion is removed. Routine observations are also reduced at graded intervals over a period of a week.

**Cardiorrhaphy** Operation is required when there is

- 1 A large sucking chest wound
2. Continued hemorrhage through the wound
- 3 A sudden relapse or a continued insidious decline after aspiration
- 4 An associated lesion, especially a large hemopneumothorax which means a significant wound of the heart or of one of the great vessels (13)

To achieve success with these lesions, all hospitals should have a set of sterile instruments and drapes always available the same set also being available for use in cardiac arrest. While the resuscitation squad is treating shock as described above a second team is preparing for thoracotomy.

**ANESTHESIA.** If the patient is moribund oxygen alone is required, but as a rule general anesthesia with cyclopropane and intubation is desirable.

**APPROACH AND TECHNIC OF OPERATION** With the patient supine the heart is approached transpleurally by means of a left fifth intercostal space incision, which is carried upward in front by transecting the fifth, fourth and third costal cartilages and corresponding intercostal muscle bundles (Fig 2) The internal mammary vessels are ligated and divided. The pericardial cavity which appears plum red and tense is opened. When the fluid and blood clot are released, hemorrhage may start afresh. Ventricular wounds are controlled by digital pressure and atrial ones by using Brock or Satinsky clamps. Blood is saved and used for autotransfusion. The heart wound is then sutured with 2-0 silk sutures on curved atraumatic needles, care being taken to underrun any branch of the coronary artery (Fig 3) In one case with an incomplete ventricular defect 4 cm in diameter direct suture was impossible and the defect was closed by suturing a free pericardial graft into place over it (17).

The pericardium is usually left open, any associated lung injury is sutured, a pleural hematoma is evacuated, and the chest wall is closed in layers over water-seal drainage.

### POSTOPERATIVE MANAGEMENT

Blood transfusion and oxygen therapy are continued, and the patient is kept on the operating table until blood pressure becomes stabilized at near normal levels. When, however hypotension persists and the patient does not quickly become conscious, recovery is unlikely because of irreversible cerebral damage.

The routine management of thoracotomy applies, and the patient rests in bed for at least three weeks.



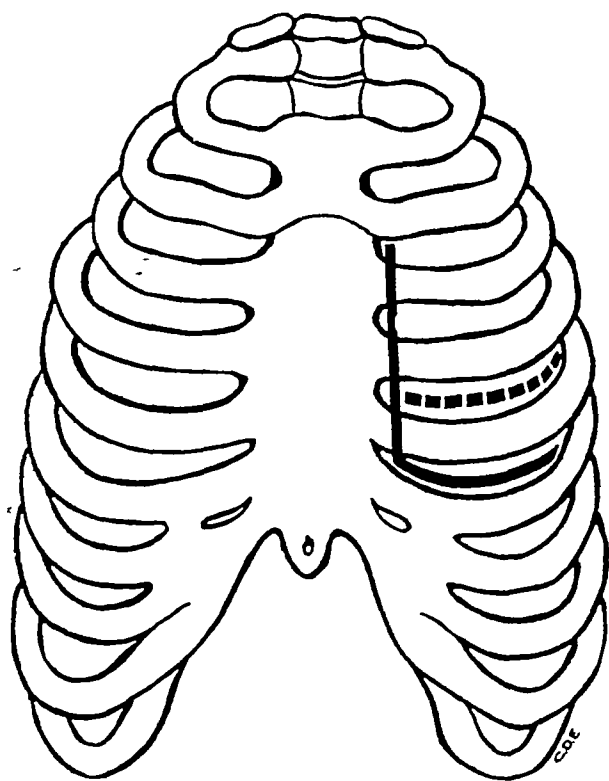


Fig 2 Diagram of operative approach for cardiorrhaphy

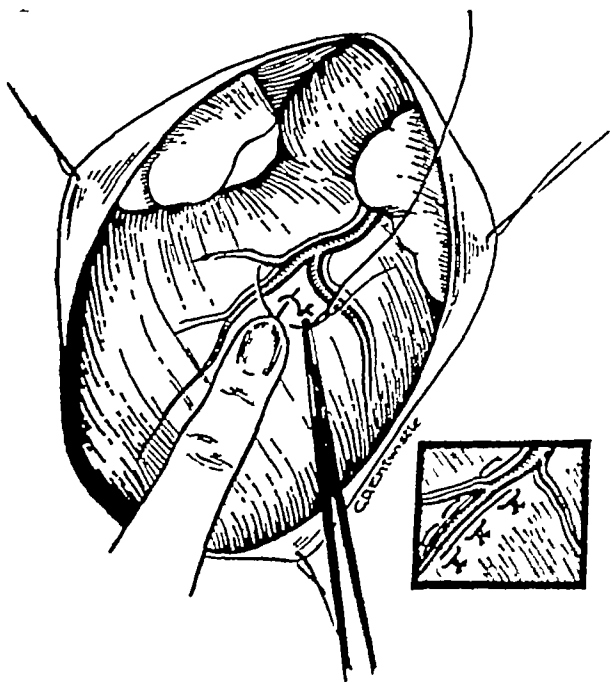


Fig 3 Technic of suturing myocardium

## POSTOPERATIVE COMPLICATIONS

As can be expected, these complications are due both to the local lesion and to the effects of the thoracotomy. Maynard and associates (5) reported *pleural effusion* in over 50 per cent of cases. On rare occasions, this effusion may proceed to *empyema*, in which event it is best treated by aspiration or closed drainage. *Pneumothorax* requires water-seal drainage and suction.

**Pericardial Effusion.** This may develop when the pericardium has been sutured either in part or completely. It is treated by careful aspiration. If infection occurs the pleura must be drained.

**Secondary Hemorrhage.** Rarely this can occur from the sutured wound with signs of return of shock and tamponade. The situation can be saved only by reopening the wound and inserting further cardiac sutures.

**Pulmonary Complications.** These complications (14) are the same as for any thoracotomy and include bronchial obstruction (a) during the operation from blood-stained mucus or vomitus and (b) after the operation from sputum retention. Careful endobronchial toilet, including bronchoscopy, is required at the end of such an operation and possibly during convalescence. If there is the least difficulty in raising sputum tracheotomy is advised (see Chapter 3).

**Infection of the Stab Wound.** Infection may occur and require local treatment.

## RESULTS OF TREATMENT

Some patients can undoubtedly recover without surgical treatment. In 1943 Blalock and Ravitch (18) reported 4 cases of which 3 were treated by aspiration, with survival of all 4. In 1949 they added (19) another 8 cases of heart wound with tamponade of which 7 had aspiration alone and 1 was treated by operation. All 8 recovered. Similarly Elkin (20) reported 18 stab wounds of which 17 were treated conservatively with a 6 per cent mortality.

Such cases however represent only a fraction of all heart wounds seen. Aspiration is effective in a small proportion of mild cases but is dangerous as a routine. Griswold and Drye (14) reporting on 108 patients in the 20-year period from 1933 to 1953 have noted an improved operability rate from 60 up to 89.5 per cent, with a decrease in total mortality from 46 to 31.5 per cent, while maintaining an operative mortality of less than 25 per cent. *The most important change in management during the 20 years had been a decrease in the extent of diagnostic procedures and a resulting decrease in preoperative time lag.* Their total figures over the 20-year period were

Admissions	108	Operative deaths	20
Total deaths	41	Not operated upon	26
Operations commenced	82	Deaths (not operated upon)	21

Further they advise that *exploratory thoracotomy is safer than aspiration as a general routine because*

- 1 The site and extent of cardiac injury can be adequately determined only by exploration
- 2 Without exploration associated injuries to other thoracic and abdominal structures may be overlooked
- 3 Intrapericardial clotting may prevent effective aspiration
- 4 Blind aspiration even via the costoxiphoid approach may prove hazard

5. Secondary hemorrhage or cardiac aneurysm may result from unsutured cardiac wounds,
- 6 Organizing pericardial clots may end in constrictive pericarditis

### CONCLUSION

All major hospital should be prepared for prompt treatment of cardiac wounds. Antishock measures and aspiration are the first steps. If there is no immediate response, then exploration with cardiorrhaphy should straightway be undertaken with the object of arresting hemorrhage, relieving tamponade, and correcting associated lesions.

### REFERENCES

- 1 Beck, C S Wounds of the heart, the technique of suture, Arch Surg, 13 205, 1926
- 2 Matas, R Surgical treatment of perforating and bleeding wounds of the chest, J A M A, 32 687, 1899
- 3 Ramsdell, E C Stab wounds of the heart, Ann, Surg, 99 141, 1934
- 4 Nelson, H Penetrating wounds of the chest A routine management based on a 5 year period of personal observation and on 5 personal cases, Arch Surg, 47 571, 1943
- 5 Maynard, A DeL, Cordice, J W V, and Naclerio, E A Penetrating wounds of the heart A report of 81 cases, Surg, Gynec, & Obst, 94 605, 1952
- 6 McKusick, V A, Kay, J H, and Isaacs, J. P Constrictive pericarditis following traumatic hemopericardium, Ann Surg, 142 97, 1955
- 7 Valle, A R War injuries of heart and mediastinum, Arch Surg, 70 398, 1955.
- 8 Davidson, J, and Fiddes, F S Prolonged activity and movement after penetrating stab wound of the heart, Brit M J 1 210, 1956
- 9 Gould, G M, and Pyle, W L Anomalies and Curiosities of Medicine, New York, Sydenham, 1936, 616
- 10 Mason, L B, Warshauer, S E, and Williams, R W Stab wound of the heart with delayed hemopericardium, J Thoracic Surg, 29 524, 1955
- 11 McGuire, C H Discussion of Elkin, D C., and Campbell, R E, Cardiac tamponade treatment by aspiration, Ann Surg, 133.623, 1951
- 12 Steffens, W Herzsteckschusse, Leipsig, George Thieme, 1936 (Quoted in ref 6)
- 13 Chamberlain, J M, Carberry, D M, and Stefko, P L Practical aspects in management of stab wounds of the heart, Am J Surg, 91 600, 1956
- 14 Griswold, R A, and Drye, J C Cardiac wounds, Ann Surg, 139 783, 1954
- 15 Crastopol, D, Goldberger, E, Marcus, R M, and Ostrone, L Wounds of the heart and pericardium, Am J Surg, 76 412, 1948
- 16 Farringer, J L and Carr, D Cardiac tamponade, Ann Surg, 141 437, 1955.
- 17 Inmon, T W, and Pollock, B E Bullet wound of left ventricle, Am Heart J. 49 459, 1955
- 18 Blalock, A, and Ravitch, M M A consideration of the nonoperative treatment of cardiac tamponade resulting from wounds of the heart, Surgery, 14 157, 1943
- 19 Ravitch, M M, and Blalock, A Aspiration of blood from the pericardium in treatment of acute cardiac tamponade after injury; further experience with report of cases, Arch Surg, 58 463, 1949
- 20 Elkin, D C, and Campbell, R E Cardiac tamponade Treatment by aspiration, Ann Surg 133 623, 1951

## PURULENT PERICARDITIS

**Introduction.** In 1819 Romero (1) of Barcelona drained a serous pericarditis between the fifth and sixth ribs, and, in 1844, Hilsmann (2) reported curing a patient with purulent pericarditis through a fourth intercostal-space incision. There after however few patients came to surgical treatment, for until 1933 Truesdale (3) collected only 152 cases from the literature. By 1941 this total had risen to 265 cases (4). As with empyema, the introduction of antibiotics has greatly lowered the incidence and severity of purulent pericarditis. Prior to 1941, Strieder (4) reported that the mortality among untreated patients was 100 per cent with pericardiectomy it fell to approximately 40 per cent, and, combined with antibiotics, it could be reduced below 30 per cent. Even in apparently moribund patients striking cures have followed operation.

## PATHOLOGY

Purulent pericarditis may occur in any age group but is most common in the first three decades. Males are more frequently affected than females (5, 7).

Purulent pericarditis is now rarely seen and is almost invariably secondary to some other infection. It is most commonly a complication of pneumococcal staphylococcal, or streptococcal pneumonia, in which cases the infection is either blood borne or a direct extension from an accompanying empyema. It may also arise by direct extension from without following precordial stab wounds, and from within following penetration of esophageal foreign bodies. Rarely it complicates thoracic operations especially those involving opening of the pericardial cavity.

Purulent pericarditis may itself be associated with a collection of air producing *pyopneumopericardium* (6). This gas may reach the pericardial sac via a stab wound, spontaneous rupture of an esophageal neoplasm, or traumatic rupture of the bronchial tree. It may further arise from such abdominal causes as a perforated peptic ulcer or a hepatic or a subphrenic abscess or following faulty pericardial aspiration or surgical drainage.

The pericardial infection causes an effusion and outpouring of fibrin which quickly lines both pericardial layers giving the typical "bread and butter" appearance. There are two major effects on the patient:

1. A generalized toxemia
2. Cardiac tamponade from embarrassment by fluid and fibrin.

Untreated the lesion rapidly proves fatal.

The frequency of the lesion in preantibiotic days is emphasized by Pyrah and Pain (7) who found acute suppurative pericarditis in 91 or 1.1 per cent, of 7,965 autopsies at Leeds from 1921 to 1931. Clinical recognition has always been difficult. Cabot (8) states that 77 per cent of all cases of pericarditis found at autopsy at the Massachusetts General Hospital were not diagnosed before the patient died. This is



Fig 1 Roentgenogram showing pericardial shadowing proved by aspiration to be pyopericardium.

confirmed by Bigger (9) in his analysis of 17 cases with acute fibrinopurulent pericarditis. While 7 frankly purulent cases were diagnosed correctly and 6 were drained, the remaining 10 were diagnosed only at autopsy. Only 4 of these 10 showed increased pericardial fluid, while in the other 6 death occurred from profound toxemia before frank pus could form in the pericardial cavity. It is noteworthy that all 4 patients with demonstrable pericardial fluid at autopsy were moribund on admission, 3 dying within 12 hours and the fourth within 24 hours of admission.

The importance of prompt diagnosis and treatment is therefore obvious.

### CLINICAL FEATURES

**Symptoms.** These may be completely masked by those of the cause. At first, the patient complains of general malaise and precordial or substernal pain. Later, as tamponade develops, dyspnea and a feeling of suffocation are added. Pressure may also cause dysphagia and cough, while irritation of the phrenic nerve may cause troublesome hiccough.

**Physical Signs.** These hold the key to diagnosis of purulent pericarditis. In addition to signs of infection with fever, tachycardia, and profuse sweating, the local findings show a greatly increased area of cardiac dullness and a characteristic pericardial friction rub. This is usually harsh, occurring during both systole and diastole, heard most loudly to the left of the sternum. When a pleuropericardial rub is present, it is usually heard more laterally, especially during inspiration. In children, there may be precordial bulging with prominent intercostal spaces.

As tamponade develops, the arterial pulse becomes faster and weaker, the cervical venous pressure higher, the area of cardiac dullness greater, and the heart sounds and pericardial rub fainter. The blood pressure similarly shows a fall in systolic and a rise in diastolic levels.

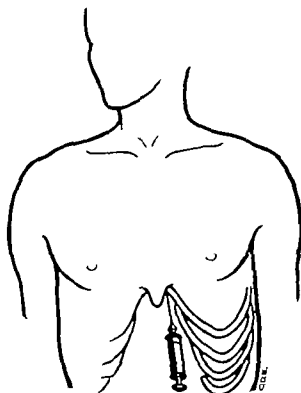


Fig. 2. Technic of aspiration of pericardium approach. With the patient semirecumbent, the angle between the xiphoid process and the left costal margin is gently infiltrated with local anesthetic, and the needle passed upward, backward, and inward into the pericardial cavity.

In the presence of pneumopericardium the area of cardiac dullness suddenly disappears and is replaced by hyperresonance. Auscultation reveals a typical metallic sound with splashing and tinkling apical sound synchronous with the heart beat. As the heart is usually pushed forward, care should be exercised in performing diagnostic aspiration.

**Investigations.** There are two—namely roentgenography and aspiration—which confirm clinical findings.

**ROENTGENOGRAPHY.** Fluoroscopy shows the enlarged pericardial sac with obliterated cardiophrenic angles and restricted cardiac movement (Fig. 1). In the presence of air a fluid level is seen, and the heart outline is clearly distinguished from the pericardium.

**ASPIRATION.** Diagnosis is confirmed by pericardial aspiration.

**Technic of Aspiration** (Fig. 2). The equipment used is the same as for pleural aspiration. The patient reclines in a semirecumbent position. The safest line of approach, through the angle between xiphoid process and left costal margin, is gently infiltrated with local anesthetic and the needle passed upward, backward and inward until the pericardial cavity is entered. Thereafter a 3-way tap and a wide bore needle (for example gauge 15 or 17) is used as being less likely to block with fibrin. A specimen of pus is sent for bacteriologic examination, and a mega of penicillin is instilled. On rare occasions, early aspiration has effected a cure (10) but the very real danger of fibrin retention rapidly leading to constrictive pericarditis as described by Bailey (11) makes this a most unsatisfactory method. Winalow and Shipley (5) consider aspiration worthless as a curative agent but agree that, by temporarily relieving severe tamponade it can tide a patient over a critical period.

**Diagnosis.** Purulent pericarditis is to be suspected from the history and physical findings and confirmed by aspirating pus, but the chief point in making this diagnosis is *to suspect its presence in all febrile conditions*. Only thus can it be diagnosed early.

### TREATMENT

The general principle governing all purulent infections applies—namely, *efficient dependent drainage at the earliest possible moment*. The technical details have altered but little since first described by Allingham (12) in 1900. Others have attempted drainage by resecting the left fourth or fifth ribs or through the sternal body, but these sites are too high, do not allow of *dependent* drainage, and are not recommended.

For *children*, Allingham advised a left subcostal approach, incising the left rectus abdominis muscle, avoiding the peritoneum, entering the cellular interval between the sternal and costal fibers of the diaphragm, and incising the tense pericardium at the lowest part of its anterior wall. As Donaldson (13) points out, this is the safest approach for *nonpurulent* effusion.

For *adults*, Donaldson advised removing the seventh and sometimes the sixth costal cartilages in order to allow for adequate digital exploration.

**Technic of Drainage.** With the patient semirecumbent, local anesthesia is infiltrated into the left fifth to seventh intercostal nerves and around the corresponding costal cartilages (Fig 3)

An oblique incision is then made over the seventh left costal cartilage, the perichondrium incised and stripped clear, and the cartilage removed. The internal mammary vessels are ligated with chromic catgut and divided. The underlying pericardium is exposed and the presence of pus confirmed by careful aspiration. A 2 to 3 cm incision is made in the pericardium, and pus at first spurts out under great tension. It is removed by suction and the pericardial cavity is irrigated with warm saline solution. A window of pericardium is next removed to provide better drainage and for histologic study. A finger is then gently insinuated and filmy loculi are broken down. Force should be avoided lest it cause myocardial laceration.

Drainage tubes are not well tolerated in the pericardial cavity, but two soft Penrose rubber-tube drains may be inserted beneath the heart and sewed to the skin. An alternative is to insert the opened end of a de Pezzer catheter just through the pericardial opening. Thereafter, pericardium and perichondrium are sutured together and absorbent dressings applied.

**Postoperative Care.** After-care is important and should be as follows:

- 1 Systemic antibiotics are continued for ten days after operation;
- 2 The dressings require frequent changing until the discharge lessens;
- 3 Daily irrigation of the tubes with an appropriate antibiotic solution in saline is advised. If fibrin formation is excessive, fibrinolytic enzymes should be used. Schweitzer (14) reporting a case, advised using a saline solution containing 100,000 units of streptokinase and 25,000 units of streptodornase;
- 4 It is important to ensure that the skin does not heal over before the pericardial infection has subsided. This is best accomplished by gentle dilatation of the opening with a sterile gloved finger,
- 5 As the pericardial cavity does not allow of completely dependent drainage when the patient is recumbent, he should be turned onto his face at regular short intervals during the day,
- 6 Iron therapy is continued as for thoracotomy. Healing is usually progressive and complete within four to six weeks.

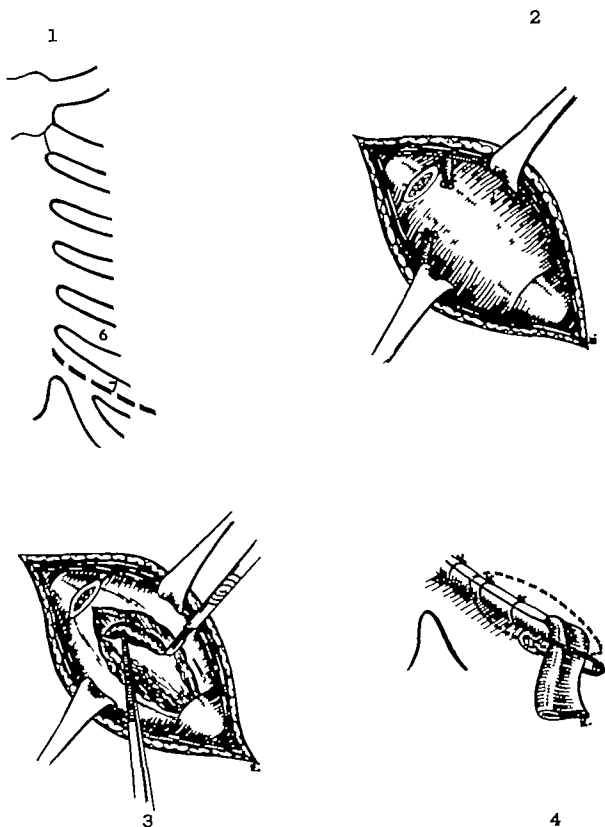


Fig. 3 Technic of drainage of purulent pericarditis. 1 Line of incision for approaching pericardium 2, Underlying pericardium exposed. 3 Incision of two to three cm. made in pericardium. 4 Pericardium and perichondrium have been sutured with drainage tube in place.



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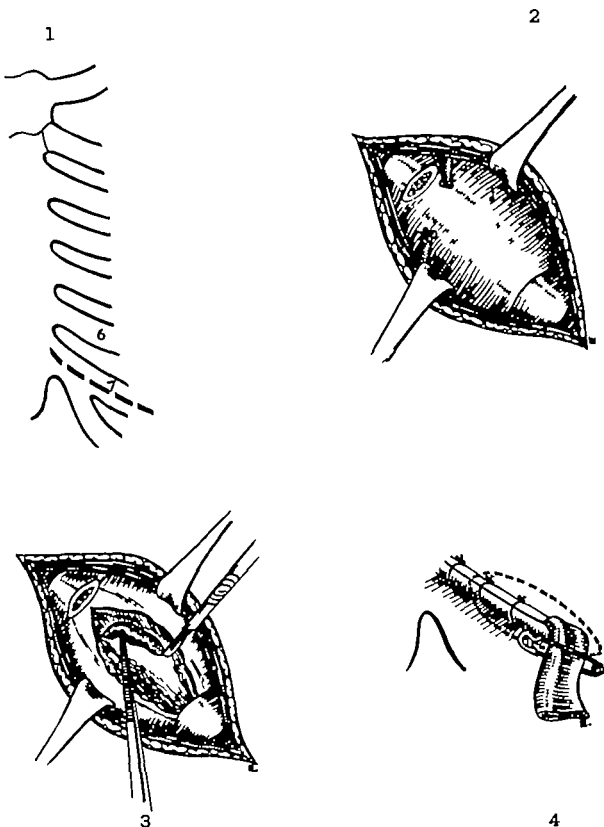


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On January 18, 1949, pericardiostomy was performed by excising the left fifth and sixth costal cartilages. When the pericardium was opened, pus spurted out under great tension.

The patient made an uninterrupted recovery and was discharged fit and with his wound healed 47 days later (Courtesy Mr. G. A. Mason.)

## COMPLICATIONS

### During Drainage.

- 1 *Cardiac arrest* may occur, but if resuscitative measures are applied promptly, recovery is possible (5). (See Chapter 24.)
- 2 If the *pleural cavity is accidentally opened*, it should be sutured and intercostal water-seal drainage established as a safeguard against empyema.
- 3 Similarly, when there is an accompanying empyema, this may be treated simply and effectively by intercostal water-seal drainage.

**Postoperative.** The most frequent postoperative complication is *loculation of pus* within the pericardial cavity. This responds to gentle digital exploration, breaking down of loculi, and further irrigation with streptokinase and streptodornase, as previously described. Winslow warns of the advisability of using a return tube, for in one patient the pericardial opening blocked during irrigation with one tube, and the resulting pressure of the trapped fluid caused fatal tamponade.

*Secondary hemorrhage* has been reported only when hard rubber tubes have been used and have eroded the cardiac blood vessels. Such a complication is therefore preventable, but if it occurs, thoracotomy and control by direct suture offer the only real hope of success.

CASE REPORT. Miss D. M., aged 19 years, on March 17, 1958, had a left lower lobectomy for bronchiectasis. That evening there were signs of severe blood loss, and x-ray films confirmed an intrathoracic hematoma. Following a four-liter blood transfusion, the wound was reopened and the hematoma evacuated. Nine days after the operation, a left staphylococcal empyema was treated by water-seal drainage. There was a positive blood culture.

On the 16th day, the patient still had a swinging temperature, a poor volume pulse of 140 per minute, a persistently low blood pressure of 80/60 mm Hg, besides signs of cardiac tamponade with increased cardiac dullness, diminished heart sounds, a basal pericardial friction rub, and prominent neck veins. X-ray films supported the diagnosis of pericardial effusion, which was proved to be purulent by aspiration. Under general anesthesia, pericardiostomy was performed by the above technique, the left fifth and sixth costal cartilages being removed. Over 200 ml. of pus were evacuated, and a soft-rubber dam drain left in situ for two days. Two weeks later, because of loculation, the sinus track was digitally explored and residual loculi broken down. She was discharged fit and well eight weeks after the lobectomy.

## CONCLUSION

Though now rare, purulent pericarditis should always be kept in mind, especially when the practitioner is seeking the answer to any obscure infection. Prompt, adequate surgical drainage offers the only chance of survival.

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- 2 Hillsmann, F. H. Cited by Alexander E. G. (see ref. 1)
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- 5 Winslow N., and Shipley A. M. Pericardiotomy for pyopericardium. Review of literature to May 1927 and report of ten new cases, *Arch. Surg.*, 15 317 1927
- 6 Meyer H. W. Pneumopyopericardium, *J. Thoracic Surg.*, 17 62, 1948.
- 7 Pyrah, L. N., and Pain, A. B. Acute suppurative pericarditis. Two cases successfully treated by operation, *Lancet*, 1 905 1933
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- 9 Bigger L. A. Suppurative pericarditis, *Ann. Surg.*, 109 763 1939
- 10 Riale E. H. Cited by Truesdale, P. E. (see ref. 3)
- 11 Bailey C. P. Surgery of the Heart, London Henry Kimpton, 1935
- 12 Ogile C., and Allingham, H. A suggestion for a method of opening the pericardial sac, founded upon a case of purulent pericarditis, *Lancet*, 1 693 1900
- 13 Donaldson, J. A. Surgical approach for incision and drainage of nonpurulent and purulent pericardial effusions, *J. Thoracic Surg.*, 12 209 1943
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## CARDIAC ARREST

This is the most sudden and serious emergency any surgeon can meet. It requires immediate action, and, unless within three minutes the hand is on the heart, manually compressing it, imitating the heart beat, and producing an adequate blood pressure and cerebral blood flow, that patient is doomed to die from cerebral anoxia. Once compression is effective, it may be required for as long as three hours before normal action is restored (1). If, however, during that period, the lungs are adequately ventilated with 100 per cent oxygen and cerebral circulation maintained by cardiac compression, the patient can recover.

**Historical Note.** The first recorded death from cardiac arrest during an operation under anesthesia occurred on Friday, January 28, 1848, when Hannah Greene was having chloroform as a prelude to the removal of a toe nail (2).

Experimentally, in 1874, the physiologist Schiff first described cardiac compression for resuscitating dogs in chloroform and ether deaths (3, 4). He clearly appreciated that the response of the heart was not from mechanical stimulation but from the filling of the coronary vessels resulting from cardiac compression. He further suggested clamping the abdominal aorta to increase peripheral resistance, found that the heart could be resuscitated even after 11½ minutes of arrest, and described assisting the circulation by further compression after the heart had recovered a feeble beat.

In human beings, Niehaus of Berne (5) first attempted cardiac compression in 1889. In 1898, Tuffier (6) reported unsuccessful transthoracic compression on a man of 24 who developed cardiac arrest in a ward. In 1900, Maag (5) had a partial success, his patient surviving 11 hours after restoration of the heart beat. In 1902, some 54 years after the initial report of death under anesthesia, Lane (7) first reported successful massage of the heart from below the diaphragm when cardiac arrest developed during appendectomy, and in 1903, Igelsrud (5) reported successful transthoracic cardiac compression after resecting the left third and fourth ribs.

By 1906, 9 successful cases had been reported (8) and, by 1909, 46 cases were collected (9). Interest, however, lagged until revived in 1941 by Bailey (10) in England and Beck (11) in the United States. In addition to manual compression, Prevost and Batelli, in 1899, introduced electric defibrillation for ventricular fibrillation, and this was first successfully used by Beck (12, 13) in 1947.

## PATHOLOGY

For practical purposes, cardiac arrest is present when the heart beat is no longer effective enough to maintain cerebral circulation.

The reported incidence lies between 1 in 804 anesthetics (14) and 1 in 4,953 anesthetics (15), with an average of 1 in 2,000 (16). It is more common in males and in the first decade of life (17).

There are two types of cardiac arrest, *cardiac asystole* and *ventricular fibrillation*. Cardiac asystole follows pure anoxia or vagal stimulation in the presence of anoxia, ventricular fibrillation follows stimulation of the heart, especially when it is anoxic.

In *cardiac asystole* the heart is toneless soft, dilated, and blue in color, with flattened coronary arteries and distended black veins. If cardiac massage is successful there is a return of tone followed by a strengthening of the beat and development of normal rhythm and appearance.

In *ventricular fibrillation* the ventricular wall is intensely active with generalized uncoordinated motion, aptly described as a "writhing bag of worms." With a favorable response to treatment, the fibrillation becomes slower, coarser, more purposeful and coordinated, until finally it is replaced by a pulse-producing beat as myocardial tone increases.

**Cause.** The fundamental cause is *myocardial anoxia* which may arise (a) in the respiratory system from inadequate ventilation or (b) in the circulatory system from hypotension or from inadequate hemoglobin for oxygen carriage. Anoxia is the greatest hazard facing any surgical patient today and it swiftly strikes the heart and brain.

#### Onset of Cardiac Arrest.

1. *During Induction of General Anesthesia* Here cardiac arrest may arise from a spasm of the vocal cords from inefficient oxygenation especially with relaxants or from profound fall of blood pressure.
2. *During Injection of Local Anesthesia* Sensitivity to local anesthesia—especially infiltration or surface application of cocaine or amethocaine—again causes profound hypotension, convulsions with laryngeal spasm, anoxia, and cardiac arrest.
3. *During the operation* cardiac arrest may arise with either a patent airway from inadequate oxygen concentration or ventilation or an airway blocked by "inhaled" vomit or sputum. (This same mechanism operates in death from drowning or suffocation.)
4. *Other Causes* Overdose of chloroform or ether causes cardiac arrest by direct action on the heart muscle. An electric shock has a similar effect. The cardiac arrest of massive hemorrhage is due to anoxia from loss of hemoglobin while that which follows an intraarterial injection of "diodone" is due to a sudden fall of blood pressure. With air embolism the coronary arteries become filled with air.

Vagal stimulation has an adverse effect, especially in the presence of heart disease and accounts for those cases seen during intubation of the trachea, bronchoscopy or cardiac catheterization of the right atrium or ventricle.

*The tendency to develop cardiac arrest is greatly increased by heart disease especially by the presence of valvular or coronary artery disease.*

### CLINICAL FEATURES

When cardiac arrest occurs after a vasovagal reflex or from drug sensitivity the onset is usually sudden but when it occurs from bronchial obstruction and anoxia there is a warning period of cardiac irregularity followed by gradual slowing of the heart beat.

With cardiac arrest, the patient is collapsed pulseless with dilated pupils, an ashen complexion, and no blood pressure. Breathing if present, is gasping in character.



## PHYSIOLOGIC APPROACH TO THE MANAGEMENT OF CARDIAC ARREST

Two fundamental steps must immediately be taken if life is to be preserved:

- 1 The airway must be cleared and artificial respiration instituted;
- 2 The hand must grasp and rhythmically compress the heart to produce an efficient blood pressure

*Lesser procedures are worthless* To inject intracardiac adrenalin, or to send for a stethoscope or a blood-pressure machine, is to play with the problem and while away precious seconds! Further, cardiac compression is the only practical way of distinguishing ventricular asystole from fibrillation

When cardiac arrest occurs while the patient is in the operating room, the heart is most readily exposed through the fifth left intercostal space. A rib spreader is inserted, the pericardium is opened, and the heart is rhythmically compressed to simulate a true, natural heart beat. The anesthetist meanwhile should intubate the patient (if the patient is not already intubated), clear the airway with suction, and artificially respire the patient with the bellows of a gas machine. If aspiration bronchoscopy is required to remove inhaled vomitus, this must be quickly carried out and the patient's lungs further artificially respired by mouth-to-bronchoscope insufflation before reinserting the endotracheal tube.

When cardiac arrest occurs in the ward or in the x-ray or out-patient departments where the services of an anesthetist are not readily available, then the heart is exposed via a midline upper abdominal incision, while any available attendant performs Sylvester-method artificial respiration until a mechanical respirator can be obtained.

If, within three minutes of the cardiac arrest, artificial respiration and manual cardiac compression are effective, the viability of the brain is at least temporarily assured, the patient has been given his only chance of survival, and the situation can be appraised. At no time can there be a halting of the process of cardiac compression and artificial respiration with 100 per cent oxygen until normal heart rhythm has been restored. As Hosler (16) summarizes, the components are:

- 1 Reestablishment of the oxygen system,
- 2 Restoration of the heart beat

### PREVENTION

*Many cases of cardiac arrest are preventable by the following measures:*

- 1 Ensure that the patient is as fit as possible for his operation, especially by checking his ventilatory capacity and the oxygen-carrying capacity of his blood. Pulmonary secretions are reduced by postural drainage and bronchoscopy, and incipient right heart failure by digitalis and mercury diuretics,
- 2 Give adequate preoperative medication, especially barbiturates, as a prelude to local anesthesia,
- 3 Treat all anesthetics and operations with the same meticulous care, even in the physically fit patient,
- 4 Always personally check all anesthetic agents, whether local or general, before administering them,
5. Use simple methods of anesthesia,

- 6 Have ready all anesthetic machine connections, airways face pieces tubes and laryngoscopes. If relaxants are being used, remember that the most thorough oxygenation allows *only three minutes* for intubation. If not successful in that time further ventilation is required before proceeding.
- 7 During the operation, the fundamental aim of the anesthetist is to see that the patient is properly oxygenated.
- 8 The surgeon must operate deftly and gently, being careful of his hemostasis and not urging too deep a plane of anesthesia to achieve relaxation. When operating on the lung, he must avoid kinking and occluding the trachea.
- 9 After the operation, the anesthetist's responsibility does not end until the patient is at least in his bed and breathing well with a good airway.

### MANAGEMENT OF CARDIAC ARREST IN THE OPERATING ROOM

*Cardiac arrest* whenever it occurs, is an unhappy situation unless there has been adequate forethought and preparation, avoidable deaths will occur. Lahey (18) has crystallized the correct attitude by stressing that this requires *team work* that it can succeed only by practice and by having everyone concerned understand what must be done and what his duty is. *This applies to every surgeon no matter what his specialty. Surgical attack on cardiac arrest should be the first operation taught any surgeon.*

Every operating room should have ready an emergency trolley containing:

- 1 Sterile instruments: scalpel, hemostats, scissors, Crafoord clamp\* and rib retractor.
- 2 Sterile syringes and needles for stimulants: neosynephrine 1 per cent solution, procaine 1 per cent, calcium chloride 10 per cent solution.
- 3 A defibrillator complete with electrodes.
- 4 Laryngoscope with blades.
- 5 A single unit bronchoscope (battery in handle) and suction apparatus.
- 6 Oxygen 100 per cent supply and bellows resuscitator.
- 7 Intravenous sets.

As soon as the emergency arises in the operating room.

- 1 The anesthetist places the patient in a steep Trendelenburg position.
- 2 He clears the airway, stops anesthesia, and continues with 100 per cent oxygen.
- 3 A nurse is instructed to call out the passing half minutes.
- 4 The surgeon prepares to expose the heart.

An incision is made in the fifth left intercostal space, the retractor inserted, the ribs spread, the heart grasped and compression commenced. Scrubbing and draping are futile. If recovery occurs antibiotics will cope with infection.

Although compression can be done for a time without opening the pericardium, sometimes with return of a beat, an effective artificial pulse can be properly achieved only by placing the hand around the heart and rhythmically contracting it 60 to 80 times per minute (Fig. 1).

\*The Crafoord aortic clamp was designed by Professor Crafoord of Stockholm.

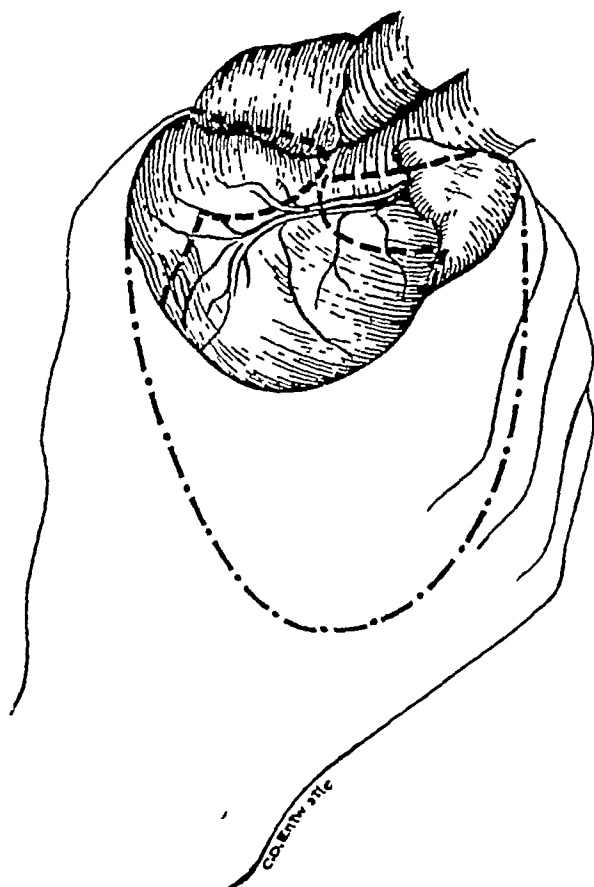


Fig 1 Diagram showing method of holding heart for effective manual compression. The apex lies in the palm of the hand, while the fingers and thumb firmly grasp and rhythmically compress the ventricles.

Peripheral resistance and therefore coronary artery filling are further increased by temporarily placing the Craford clamp on the descending thoracic aorta for five minutes at a time. Venous return is increased by having an assistant raise the patient's legs 90 degrees, thus producing in effect an autotransfusion of approximately 600 ml of blood. Effective compression is shown by a pulse at the patient's wrist, recording of a blood pressure of over 80 mm Hg, and by the development, in the patient's eye, of a small pupil that will often react to light. When there has been blood loss, radical arterial transfusion is commenced.

Relays of assistants are called to take over the cardiac compression since, when effective, this maneuver is physically exhausting.

The prime cause of failure to achieve recovery is simply that the surgeon, his assistant or the anesthetist fail to appreciate the true significance of a pulseless patient *quickly enough* and fail to get a hand onto the heart *within that vital three-minute period*. Though a heart beat may be temporarily restored *after* three minutes of cardiac arrest, the brain cannot be expected to recover, cerebral edema develops, and the patient will die, usually within 24 hours.

**Return of Cardiac Action.** Whether the heart be in asystole or ventricular fibrillation, a normal heart beat may occasionally be restored as soon as rhythmic normal compression achieves cardiac oxygenation. If the compression fails to do so, a series of drugs may be injected into the left ventricle.

*With asystole*, the aim is to increase cardiac tone and initiate sinus rhythm. Milstein (4) advises using a dose of 5 to 10 ml of 1 in 10,000 adrenalin solution if the heart is well oxygenated. Others use epinephrine 1 in 1,000, commencing with 0.5

and increasing even up to 5 ml. With cardiac anoxia, adrenalin must be avoided as it will precipitate ventricular fibrillation. Calcium chloride 5 to 10 ml of a 10 per cent solution may add tone to a flabby myocardium (19)

*With ventricular fibrillation* if cardiac tone has not improved with massage Milstein advises encouraging this by an injection of adrenalin. Should this fail, an injection of 50 to 200 mg of procaine hydrochloride may achieve the desired result. As procaine and adrenalin are antagonistic in their effect on the heart, in the absence of an electric defibrillator, alternating injections of procaine and adrenalin may be used, at five minute intervals

*The treatment of choice however is electric defibrillation* The underlying principle is to produce a current of sufficient strength and duration to cause a simultaneous contraction of all components of ventricular muscle. All the ventricular muscle then becomes relatively refractory at the same time so that a second stimulus arriving during this refractory period can produce a coordinated beat.

The large electrodes 7 to 8 cm. in diameter are placed one on each side of the ventricles and a single shock of lowest time and voltage is given. This may cause asystole for 10 to 15 seconds after which a regular beat may return.

When one shock fails to halt fibrillation, O'Reilly and Ohlke (20) advise a series of five shocks at half-second intervals. Manual compression is immediately reapplied, and defibrillation reattempted with increasing duration of the electric current. If that also is not sufficient, the voltage is increased to 220 volts and the same process repeated. Failure can be followed by further treatment with procaine and adrenalin. When asystole persists continued massage may shortly restore a normal beat.

Milstein found that procaine hydrochloride 50 to 200 mg was effective in only 9 of 23 episodes of ventricular fibrillation in which it was used. Electric defibrillation was effective in only 6 out of 14 episodes

#### TIME TO PERSIST

Although ventricular fibrillation may be *immediately* corrected by normal compression, it more usually takes 5 to 15 minutes. In some instances however despite all endeavors it continues. How long, therefore should one persist with manual compression?

Lahey and Ruzicka (2) described a patient on whom, because of temporary return of cardiac action massage was carried out for 2 hours and 45 minutes before abandoning resuscitation.

Adams (21) in 1954 reported cardiac arrest during division of a patent ductus arteriosus in an 11 year-old boy. While constantly maintaining cardiac massage at the rate of 60 to 70 compressions per minute with the patient in steep head-down position, the following was done. The heart was flicked with the finger it was allowed to fill with blood to dilatation, and then squeezed vigorously. 0.5 ml of 1:1000 neosynephrine mixed with 9.5 ml of 1 per cent procaine hydrochloride was injected into the left ventricle. electrical defibrillation was tried about 20 separate times with variable voltage and amperage. 10 ml of procaine hydrochloride solution (1 per cent) was injected into the right ventricle. 100 mg of Pronestyl hydrochloride was given intravenously and 10 ml. of 1 per cent procaine hydrochloride was injected into the right ventricle. After 90 minutes, a visible sinus rhythm appeared in the left auricle and persisted but was not transmitted to the fibrillating ventricles. At the

end of 110 minutes, normal ventricular rhythm returned and the patient recovered

Milstein (4) records a case that had 9 episodes of ventricular fibrillation requiring repeated doses of procaine and calcium chloride as well as attempted electric defibrillation before a normal heart beat was restored in 123 minutes.

Sometimes unduly vigorous massage may perforate the heart wall. Even this is not irrecoverable, for, in a case described by Haight and Sloan (1), a right ventricular laceration through a previously infarcted area was partially controlled by packs while the other hand massaged. With greatest difficulty, a running suture was placed, and after 30 minutes the bleeding was controlled. Despite previous resuscitative efforts, success was achieved only when the massage occurred synchronously with the heart's own weak contractions. Function was restored after 3 hours and 15 minutes of cardiac massage.

It is, therefore, quite obvious that the surgeon has a heavy responsibility to persist with his efforts until all chances of resuscitation and recovery are exhausted, and that ends only some three hours after the initial arrest has occurred. *So long as massage produces a good peripheral pulse and the pupils remain constricted and react to light, efforts should not be abandoned.*

The following four personal cases illustrate some of the points already enumerated.

**CASE 1** Mr. A. A., age 52, was admitted as an emergency on July 21, 1953 with right hydropneumothorax from spontaneous perforation of the esophagus. This was drained by water-seal drainage without much re-expansion of the right lung.

Following intubation with a cuffed endotracheal tube, inflation of the lung proved difficult. The patient became pulseless. He was immediately taken into the operating room, the right chest was opened by an intercostal incision, and cardiac compression of the motionless heart was commenced. The endotracheal tube was withdrawn a little, and, following 10 compressions, a normal pulse returned. The operation was then successfully concluded.

*Comment:* This asystole was asphyxial in origin. The cardiac arrest was probably due, first, to the endotracheal tube being inserted too far and occluding the left main bronchus, and, second, to fibrin over the right lung limiting its ability to be aerated. Because the arrest was promptly recognized and treated, there were no sequelae.

**CASE 2** On July 28, 1953, Miss G. C., age 52, who had mitral stenosis with normal rhythm but with pulmonary hypertension, was prepared for mitral valvulotomy. Anesthesia was induced in the anesthetic room with 0.5 gm thiopentone and 18 mg of tubarine. A cuffed endotracheal tube was inserted, and the patient became pulseless.

The author was called from a nearby operating room, confirmed the pulseless state, gave an intracardiac injection of neosynephrine 1 ml with no response, and followed this with a midline upper abdominal incision and cardiac compression. Here, the hand could easily ride up behind the pulseless heart, allowing rhythmic compression between hand and sternum without opening the diaphragm. With the anesthetist giving pure oxygen and with effective compression, the patient's color improved. Within 30 seconds, sporadic beats occurred, and these were rapidly followed by a return of normal rhythm. Swallowing began within 40 minutes, her pupils reacted to light an hour after the incident, and her corneal reflexes returned within 2½ hours. There was a slow but steady rise of blood pressure, she became conscious 16 hours after onset, and thoroughly comatose within two days.

Six weeks later mitral valvotomy was successfully performed.

*Comment* Here the cause was probably vagal in origin. The initial injection of neosynephrine wasted a precious minute and considerably lengthened the time for return of consciousness. The abdominal route was felt to be reasonable in the anesthetic room and, in this case, proved satisfactory.

**CASE 3** On June 23, 1955, Mr. W. H. was being prepared for right thoracotomy to remove a pulmonary neoplasm. During intubation, the neoplasm bled freely into the bronchial tree and the patient rapidly became cyanosed. The author was summoned, commenced aspiration bronchoscopy and, as the patient was now pulseless, the surgical resident commenced a left thoracotomy. It was quickly realized, however, that the left lung alone was functioning and that a left pneumothorax would embarrass its action.

The heart was therefore exposed through a midline upper abdominal incision, followed by ventral detachment of the diaphragm. There was complete asystole. Cardiac compression was commenced at approximately 60 beats per minute. Within a minute, heart beats returned, first one, then three, six and nine—separated by periods of asystole. Thereafter—without further compression—normal rhythm was restored. The wounds were closed and recovery was uneventful. However, the patient declined further surgical treatment, and died, three months later, from his neoplasm.

*Comment* The cause in this case was asphyxia from endobronchial blood clot. The neoplasm had occluded the right lung and, as intubation was impossible without inviting further hemorrhage, left transthoracic compression with resulting pneumothorax was out of the question. The only alternative was the transabdominal approach which proved satisfactory.

**CASE 4** Mrs. L., at the moment of commissurotomy for mitral stenosis, commenced ventricular fibrillation. The index finger of the operator's right hand was quickly removed from the left atrium, the auricular opening was clamped, and cardiac compression was commenced. One ml. of adrenalin was injected into the heart, followed by procaine amide. Following the tachycardia, normal rhythm was restored, and recovery was uneventful.

*Comment* This case illustrates how defibrillation can be successfully undertaken by drugs when no defibrillator is available.

## RECOVERY

This may be complete, partial, or absent. Those with partial recovery have full heart and lung function but little return of cerebral activity and they may linger on in chronic dementia even for years. The severity of neurologic complications is directly proportional to the delay between cardiac arrest and effective restoration of circulation.

## AFTER-CARE

The following points require utmost attention:

1. The blood pressure must be maintained at over 80 mg. Hg, if necessary with a noradrenalin drip infusion. Oxygen is continued via nasal catheters.
2. After asystole, atropine sulfate 0.43 mg. is injected intravenously. With tachycardia, the patient should be rapidly digitalized with digoxin. After fibrillation, Hosler advises an intravenous solution of 20 per cent dextrose and 3 per cent

saline to counteract increase of potassium ions in the blood stream and accompanying arrhythmias,

- 3 The patient remains in the operating room until spontaneous respiration returns, and the pulse rate, blood pressure, and electrocardiograph readings are satisfactory. If respirations are labored, a mechanical respirator should be used. A special nurse is required from the start,
- 4 When all is well, the patient is moved to a recovery room. Pharyngeal and intratracheal secretions are regularly aspirated. A tracheotomy set is made ready lest tracheal suction prove inadequate. When respirations are vigorous, the bronchi are finally sucked clean and the endotracheal tube is removed,
5. If return of consciousness is delayed, a fine stomach tube is passed through a nostril and a fluid diet given. The bladder is watched for retention, and a strict fluid balance is kept. Agents to counteract cerebral edema have seldom been successful (22)

### RESULTS

Stephenson and associates (17) in 1953, reviewing 1,200 cases of cardiac arrest, found that the heart beat was restarted in 56 per cent of all cases and that the permanent survival rate was 28 per cent. The great majority of patients who die after the heart is restarted expire within the first 24 hours.

As 14 per cent of cases occurred outside the operating room (and 17 per cent of these were permanently resuscitated), a hospital plan of action must be prepared for meeting this emergency at any point in the hospital. *Of all successes, 94 per cent were massaged within the first four minutes.*

In a recent study of a 30-year period of operating-room deaths at Massachusetts General Hospital, Briggs (23) showed that there had been an absolute increase in the incidence of this emergency in recent years. On analysis, this recorded increase was due to a wider awareness and diagnosis of the problem, and to the increased number of surgical procedures carried out in elderly and decrepit patients. In that period, cardiac arrest was shown to have had a fivefold increase in patients with heart disease, twentyfold in the elderly and thirtyfold in poor-risk patients. Other contributing factors were deepening anesthesia, hypoxia, reflex phenomena, and improper choice or management of anesthetic. While, during the last 10 years, the recovery rate in cardiac arrest was only 37 per cent, during the last 5 years it has risen to 50 per cent.

### CARDIAC ARREST OUTSIDE THE OPERATING ROOM

The possibility of meeting cardiac arrest other than in the operating room must be seriously faced by all medical practitioners, for cardiac massage offers a hope of life.

*In the ward*, the author has had the problem arise in one of the following examples.

- 1 A patient collapsed and died while his chest was being massaged. Autopsy failed to reveal air embolism, and the cause of death was of unknown nature.
- 2 Two weeks after an uncomplicated repair of a hernia, a patient in her bed collapsed and died. Resuscitation was considered, but time was too short. Autopsy

embolism blocking one branch of the pulmonary artery to the left lower lobe. Presumably the embolus had caused a fatal ventricular fibrillation.

In both cases a doctor was present, and the author strongly feels that had the patient's abdomen and diaphragm been promptly opened to allow manual compression with the ward nurse applying Sylvester type artificial respiration, both patients could have been at least kept "safe" if not successfully resuscitated until the arrival of the resuscitation squad.

- 3 On a third occasion, cardiac arrest occurred while a patient was returning to the ward following excision of a mediastinal dermoid cyst. Manual compression was tried. Normal heart rhythm returned at once and the ward nurse applied artificial respiration until the resuscitation squad arrived 10 minutes later when the patient was intubated and respired with a respirator. There had been some initial delay in performing the cardiac massage and the patient died two hours later. This case did, however, establish that, in such circumstances, resuscitation is both possible and practical and that, when this emergency does arise if immediate action can be taken, at least some of these patients will recover.

The approach to be used varies with the place and circumstances, and already successes have been reported.

Hosler (16) tells how a 65 year-old physician collapsed when leaving a Cleveland hospital. Two clear-thinking surgeons behind him carried him to a nearby accident room, ripped off his shirt and within two minutes massaged his heart and ventilated his lungs. The heart was in coarse fibrillation, and after several electric shocks, it returned to a coordinated beat. He was successfully resuscitated without even removing the rest of his clothing.

Brown and associates (24) reported how a man age 34 while developing x ray films collapsed from cardiac arrest on the dark room floor. In this case a penknife thoracotomy was done and cardiac massage commenced. He was later carried through the x ray "maze" to a surgical table and recovered a normal heart beat two and a quarter hours later.

Finally by the same token the author feels that when he is summoned to a case of collapse from electrical shock with cardiac arrest, or to a case of drowning, and, by luck, is present within four minutes, there is everything to gain and nothing to lose by the immediate application of cardiac massage preferably transabdominal approaching the pericardium by retracting the central tendon of the diaphragm. Others present can then perform Sylvester artificial respiration.

## CONCLUSION

The sooner it is widely appreciated that in cases of cardiac arrest, artificial respiration alone is a forlorn hope and treats only half of the dual problem and the sooner there is an attitude of mind to combine artificial respiration with manual pumping of the heart, the sooner more lives will be saved.

## REFERENCES

- 1 Haight, C., and Slown, H. Successful cardiac resuscitation despite perforation of the heart during massage, *Ann. Surg.*, 141:240, 1955.
- 2 Lahey F. H., and Ruzicka, E. R. Experiences with cardiac arrest, *Surg., Gynec. & Obst.*, 90:108, 1950.



- 3 Schiff, M *Recueil des Mémoires physiologique*, Lausanne, 1874 (Quoted by Milstein, B B , in ref 4 )
- 4 Milstein, B B Cardiac arrest and resuscitation, *Ann Roy Coll Surgeons*, England, 19 69, 1956
- 5 Barber, R. F , and Madden, J L *Historical aspects of cardiac resuscitation*, *Am J Surg* , 70 135, 1945
- 6 Tuffier, T and Hallion, L *De la compression rythmée du coeur dans la syncope cardiaque par embolie*, *Bull et Mém Soc Chir. de Paris*, 24 937, 1898
- 7 Starling, E H , and Lane, W A. Report of communication to Society of Anesthetists, *Lancet*, 2 1397, 1902
- 8 Green, T A , *Heart massage as a means of restoration in cases of apparent sudden death*, with a synopsis of 40 cases, *Lancet*, 2 1708, 1906
- 9 Von Caekovic, M *Ueber directe Massage des Herzens als Mittel zur Wiederbelebung*, *Arch f klin Chir* , 88 917, 1909
- 10 Bailey, H Cardiac massage for impending death under anesthesia, *Brit. M J* , 2 84, 1941
- 11 Beck, C S Resuscitation for cardiac standstill and ventricular fibrillation occurring during operation, *Am J Surg* , 54 273, 1941
- 12 Beck, C S , Pritchard, W H , and Feil, H S Ventricular fibrillation of long duration abolished by electric shock, *J A M A*. 135 985, 1947
- 13 Prevost, J. L , and Batelli, F *Sur quelques effets des décharges électriques sur le coeur des mammifères*, *C R Acad Sci* , Paris, 129 1267, 1899
- 14 Gillespie, N A Death during anesthesia, *Brit J Anaesth* , 19 1, 1944
- 15 Heckel, E , and Fell, E H Cardiac arrest during surgical operations Causes, prevention and actual management, *S Clin. North America*, 35 243, 1955
- 16 Hosler, R M Six years' experience with the Cleveland Cardiac Resuscitation Course, *A M A Arch Surg* , 73·813, 1956
- 17 Stephenson, H E , Reid, L C , and Hinton, J W Some common denominations in 1,200 cases of cardiac arrest, *Ann Surg* , 137 731, 1953
- 18 Lahey, F. H In discussion of reference 17
- 19 Kay, J H , and Blalock, A The use of calcium chloride in the treatment of cardiac arrest in patients, *Surg , Gynec , & Obst.*, 93 97, 1951
- 20 O'Reilly, K. S , and Ohlke, R F Cardiac arrest, causation and management, *Canad M A.J* , 75 202, 1956
- 21 Adams, R in discussion of Sealy, W C , Young, W G., and Harris, J S Studies on cardiac arrest The relationship of hypercapnia to ventricular fibrillation, *J Thoracic Surg* , 28·461, 1954.
- 22 Cole, F. The use of human serum albumin in cerebral edema following cardiac arrest, report of a case, *J A M A* , 147.1563, 1951.
- 23 Briggs, B D , Sheldon, D B and Beecher, H. K. Cardiac arrest. Study of a thirty-year period of operating room deaths at Massachusetts General Hospital, 1925–1954, *J A M A* , 160·1439, 1956
- 24 Brown, C D , Knudson, J , and Schroeder, G F Cardiac arrest at work—penknife thoracotomy with recovery, *J.A M A* , 163 352, 1957

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